THE PLANT DISEASE REPORTER

Issued By

THE EPIDEMIOLOGY UNIT

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 38 Number 7

July 15, 1954

LIBRARY
CURRENT SERIAL RECORD
AUG 2 - 1954
DEPARTMENT OF AGRICULTURE

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Plant Disease Epidemics and Identification Section serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

Issued by

THE EPIDEMIOLOGY UNIT
PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Horticultural Crops Research Branch — Plant Industry Station, Beltsville, Maryland
Volume 38 — July 15, 1954 — Number 7

IN THIS ISSUE

Certain microorganisms inhibit the growth of Endothia parasitica in culture, according to MIHAILO KRSTIĆ, page 427.

ARTHUR R. ENGLISH and G. VanHALSEMA report that a combination of streptomycin and Terramycin slows up the emergence of resistant strains of Xanthomonas and Erwinia, page 429.

R. S. KIRBY reports extremely good control of apple fire blight with antibiotics under epidemic conditions, page 432.

Results of field trials using thiuram polysulfides against several pathogens are reported by SAUL RICH and JAMES G. HORSFALL, page 434.

W. R. WRIGHT, et al., report Diplodina rot in peaches from South Carolina, page 436.

Occurrence of Verticillium wilt of sweet cherry in California is reported by STEPHEN WILHELM, et al., page 438.

Virus Diseases: The Tristeza virus is one of the factors in poor growth of satsuma oranges in Texas, according to tests reported by EDWARD O. OLSON and JAMES R. McDONALD, page 439. JOHN R. COLE describes an abnormal condition of pecan showing characteristics of both bunch and rosette, observed in New Mexico, page 443. W. H. SILL, Jr., et al., conclude that wind rubbing is a minor factor in transmission of the Wheat Streak-Mosaic Virus, page 445. The Crimson Rambler morning glory is a good indicator plant for the sweetpotato Feathery Mottle Virus, according to transmission experiments reported by RAYMON E. WEBB, page 448. In discussing the virus disease complex of canning peas in the Pacific Northwest F. P. McWHORTER reports evidence indicating that mingling of viruses may extend the host range of a single virus, page 453. A. A. COOK reports association of several strains of Virus X with "freckles", observed in potatoes in South Dakota, page 458.

Use of vermiculite increases effectiveness of potato scab inoculations according to G. V. C. HOUGHLAND and L. C. CASH, page 460.

N. J. WHITNEY reports violet root on celery in Ontario, page 462.

W. B. NEUBERG, et al., describe a wrinkled seed coat defect in green lima beans for freezing, page 464.

L. A. BRINKERHOFF, with others, reports tests with chemicals for the control of cotton seedling diseases: field tests, page 467; greenhouse tests, page 476.

THOMAS E. SUMMERS reports the existence of physiologic races in Colletotrichum hibisci on kenaf, page 483.

Screening tests for fungicides to control black stripe of the Hevea rubbertree are reported by J. B. CARPENTER, page 487; and J. B. CARPENTER and J. A. STEVENSON describe a secondary leaf spot, page 494.

DON C. NORTON reports a Fusarium root rot of guayule, found in Texas during recent years, page 500.

G. B. LUCAS and F. A. TODD discuss seedling and transplant resistance to black shank in some tobacco varieties, page 502.

A race of crown rust different from any previously seen in North America appeared in 1953 and 1954 collections according to M. D. SIMONS, page 505.

Diseases of pearl millet occurring in Georgia are described by E. S. LUTTRELL, page 507.

J. A. PARMELEE reports Curvularia on gladiolus in Canada, page 515.

Ceresan M was the most effective of fungicides used for gladiolus corm treatment to prevent dry rot in storage according to FRANK A. HAASIS, page 518.

Progress report of the Committee on Seed and Plant Material Certification, page 521.

Unusual records and brief notes, page 529: Lime paste for reducing loss from sunburning of watermelon, by G. K. PARRIS; Root diseases of cotton in Mexico and California, by JOHN T. PRESLEY; Keeping deciduous oaks in leaf during the winter, by BERT M. ZUCKERMAN and P. F. HOFFMAN; southern blight on Philodendron in California, by ALEX M. FRENCH and CARL W. NICHOLS.

Announcements, page 531: "Maneb" for manganese ethylenebisdithiocarbamate; Results of 1953 fungicide tests.

May weather, page 532.

NOTE ON THE SUSCEPTIBILITY OF THE CHESTNUT-BLIGHT FUNGUS TOWARDS SOME MICROORGANISMS IN CULTURES

Mihailo Krstić[1]

Some microorganisms have shown an inhibitory activity when grown in vitro with Endothia parasitica (Murr.) P. J. & H. W. Anders., isolated from European chestnut (Castanea sativa Mill.) in Yugoslavia. The most obvious effects were noticed when a strain of Bacillus subtilis Cohn emend. Prazmowski (No. A2) and Penicillium rubrum Stoll were grown with E. parasitica on potato-dextrose agar in Petri dishes. A permanent zone of inhibition from 3-6 mm. and free of any mycelial growth was established in the case of P. rubrum, while a temporary free zone, extending from 4-8 mm., was noticed in the case of B. subtilis. The mycelium of P. rubrum spread sometimes over the surface occupied by the parasitic fungus. Poorer mycelial growth was noticed in proximity to B. subtilis colonies. A more rapid formation of pycnidia took place on the margin that marked the limit of normal growth of Endothia.

A similar effect of B. subtilis (strain A2) and of an undetermined Penicillium sp. has been observed with the American strain of Endothia parasitica in preliminary experiments carried out at the Plant Industry Station, Beltsville, Maryland. On potato-dextrose agar plate the definitely inhibited zone amounted to 5-6 mm. in the case of Penicillium, and a temporary zone of 6-7 mm. in the case of B. subtilis (Figs. 1 and 2). A secondary mycelial growth of E. parasitica towards B. subtilis colonies was slower and in some cases did not reach these colonies.

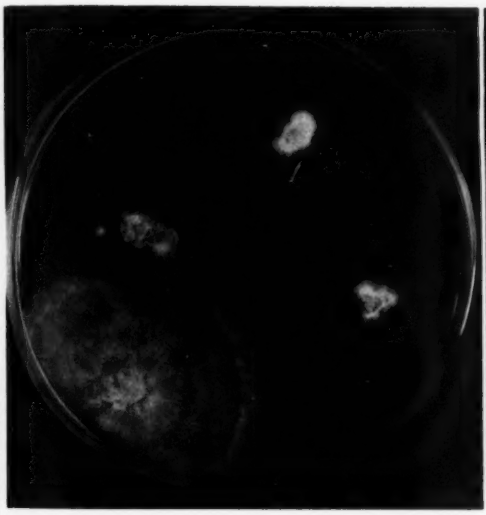


FIGURE 1. Growth of American strain of Endothia parasitica inhibited by Penicillium sp. Culture of E. parasitica is 7 days old; Penicillium sp. is 5 days old.

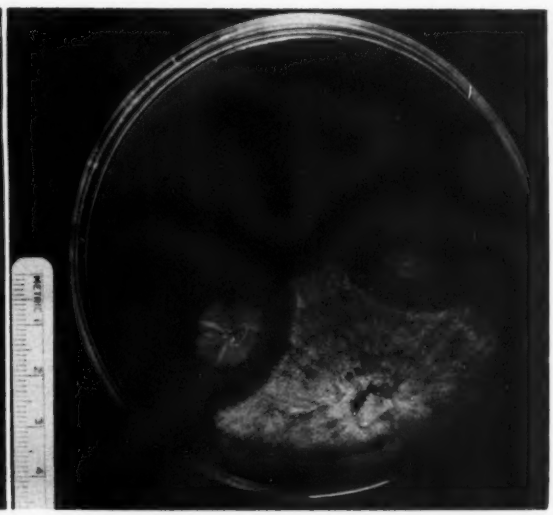


FIGURE 2. Growth of American strain of Endothia parasitica retarded by Bacillus subtilis colonies. Culture of E. parasitica is 7 days old; Bacillus subtilis is 5 days old. Discolored zone between the two organisms is due to a chemical change of medium.

Using the dilution method, it was demonstrated that sterilized metabolic liquids, derived fromfour- to ten-day-old cultures of P. rubrum grown in potato-dextrose broth, inhibit the germination of pycnidiospores and the mycelial growth of E. parasitica (Yugoslav strain) in dilutions of 1:1.5 - 1:3. Germination and growth were retarded in dilutions up to 1:5. The sterilized culture fluids of B. subtilis retard the germination of pycnidiospores and the growth of the mycelium itself in 1:2 dilution, inducing at the same time a weakened production of pycnidia on

[1]Forest Pathologist, Forest Research Institute, Belgrade, Yugoslavia.

the liquid surface. When filtered through a Seitz filter, the metabolic fluids of both P. rubrum and B. subtilis became more active and completely inhibited the germination of pycnidiospores in 1:3 - 1:7 dilution of P. rubrum and 1:1 of B. subtilis.

Among other microorganisms examined, Aspergillus niger v. Tiegh. showed a slighter inhibitory effect. An undetermined Gram-positive, sporulating bacillus, producing slimy colonies on potato-dextrose agar and possessing a bacteriolytic property against Staphylococcus aureus Rosenbach, had a temporary effect against E. parasitica. The activity of this bacillus appeared changeable but in some cases was more evident than that of P. rubrum and B. subtilis. The zones of temporary inhibition in Petri dishes were found to be from 0.5 - 6.5 mm. Pycnidia were formed around the bacillus colonies. By sterilization the culture fluids lost their fungistatic activity and the mycelium of E. parasitica became capable of growing normally in undiluted cultures of this bacillus. If filtered through a Seitz, a weak inhibitory effect (1:0.5) was noticed in eight day-old bacillus cultures.

Conclusions

These experiments demonstrate that E. parasitica possesses a certain amount of sensitivity towards some common saprophytic microorganisms, especially B. subtilis and Penicillium species. Its reaction to a number of other common bacteria and molds has not yet been investigated in this manner. It seems desirable, however, to conduct further experiments to study the role of these agents in inhibiting the spread of E. parasitica under natural conditions.

FOREST RESEARCH INSTITUTE, BELGRADE, YUGOSLAVIA

A NOTE ON THE DELAY IN THE EMERGENCE OF RESISTANT XANTHOMONAS AND ERWINIA STRAINS BY THE USE OF STREPTOMYCIN PLUS TERRAMYCIN COMBINATIONS

Arthur R. English and G. VanHalsema[1]

It has been well documented that the development of resistance in various human pathogenic microorganisms to single antibiotics may be retarded by the use of antibiotic combinations (1, 2, etc.). Since single antibiotics are now receiving field trials as sprays against selected plant diseases, the possibility exists that comparable resistance phenomena might occur among the various microorganisms pathogenic for plants. Perhaps this would be particularly likely in plant diseases which require spraying at regular short time intervals, e.g., bacterial spot which is sprayed every five days. Therefore, these in vitro studies were undertaken to determine whether or not the use of an antibiotic combination of streptomycin plus Terramycin®[2] as compared with streptomycin alone would effectively delay the emergence of resistant Xanthomonas and Erwinia strains.

The streptomycin and Terramycin stock solutions used were prepared from the commercial products of Chas. Pfizer and Co., Inc. Erwinia amylovora strains #3 and #4 were originally received from the University of Missouri. The Xanthomonas vesicatoria strain, from the University of Delaware Experiment Station, was a recent isolate from pepper plants infected with bacterial spot. Resistant strains of these microorganisms were developed in vitro by the use of a serial transfer procedure which involves the exposure of the organism to progressively increasing concentrations of the antibiotic or antibiotic combination in nutrient broth (3). The rate of emergence of resistance of the microorganism to streptomycin was compared to the rate observed with a streptomycin plus 1% Terramycin or a streptomycin plus 10% Terramycin combination.

The data that are presented in the accompanying graphs clearly indicate that a combination of streptomycin plus Terramycin is much superior to the use of streptomycin alone in delaying the emergence of resistant strains of these microorganisms. After only 13 transfers of the two Erwinia strains in the presence of streptomycin alone, resistance to that antibiotic increased by 250 to 500 times. After the same number of transfers in the presence of a combination of streptomycin plus 10% Terramycin, the resistance of strain #3 had increased only two times while the resistance of strain #4 had increased only five times. The streptomycin plus 1% Terramycin combination was also very effective in delaying the rate of emergence of resistant Erwinia strains although not quite so efficient as the use of streptomycin plus 10% Terramycin.

After only nine transfers in the presence of streptomycin, Xanthomonas vesicatoria could withstand at least 500 mcg/ml of this antibiotic. However, when the combination of streptomycin plus 10% Terramycin was used, this strain could withstand only a total concentration of 10 mcg/ml of both antibiotics.

Summary

It has been shown in vitro that a combination of streptomycin and Terramycin is effective in delaying the rate of emergence of resistant strains of Erwinia amylovora and Xanthomonas vesicatoria.

Literature Cited

1. Wright, S. S., E. M. Purcell, C. Wilcox, M. K. Broderick and M. Finland. 1953. Antibiotic combinations and resistance to antibiotics. Jour. Lab. & Clinc. Med. 42: 877.
2. Jawetz, E. 1953-1954. Combined antibiotic action: Experimental and Clinical. Antibiotics Annual, 41, 1953-1954. Published by Medical Encyclopedia, Inc., New York City.
3. English, A. R., and P. C. Gelwicks. 1951. The in vitro microbial resistance pattern to Terramycin. Antibiotics and Chemotherapy 1: 118.

[1]Bacteriology Laboratory of Chas. Pfizer and Co., Inc. Brooklyn, New York.
[2]®Terramycin is the trademark of Chas. Pfizer & Co., Inc. for the antibiotic, oxytetracycline.

FIGURE 1

DEVELOPMENT OF RESISTANCE OF ERWINIA AMYLOVORA STRAINS TO STREPTOMYCIN, TERRAMYCIN, AND COMBINATIONS THEREOF.

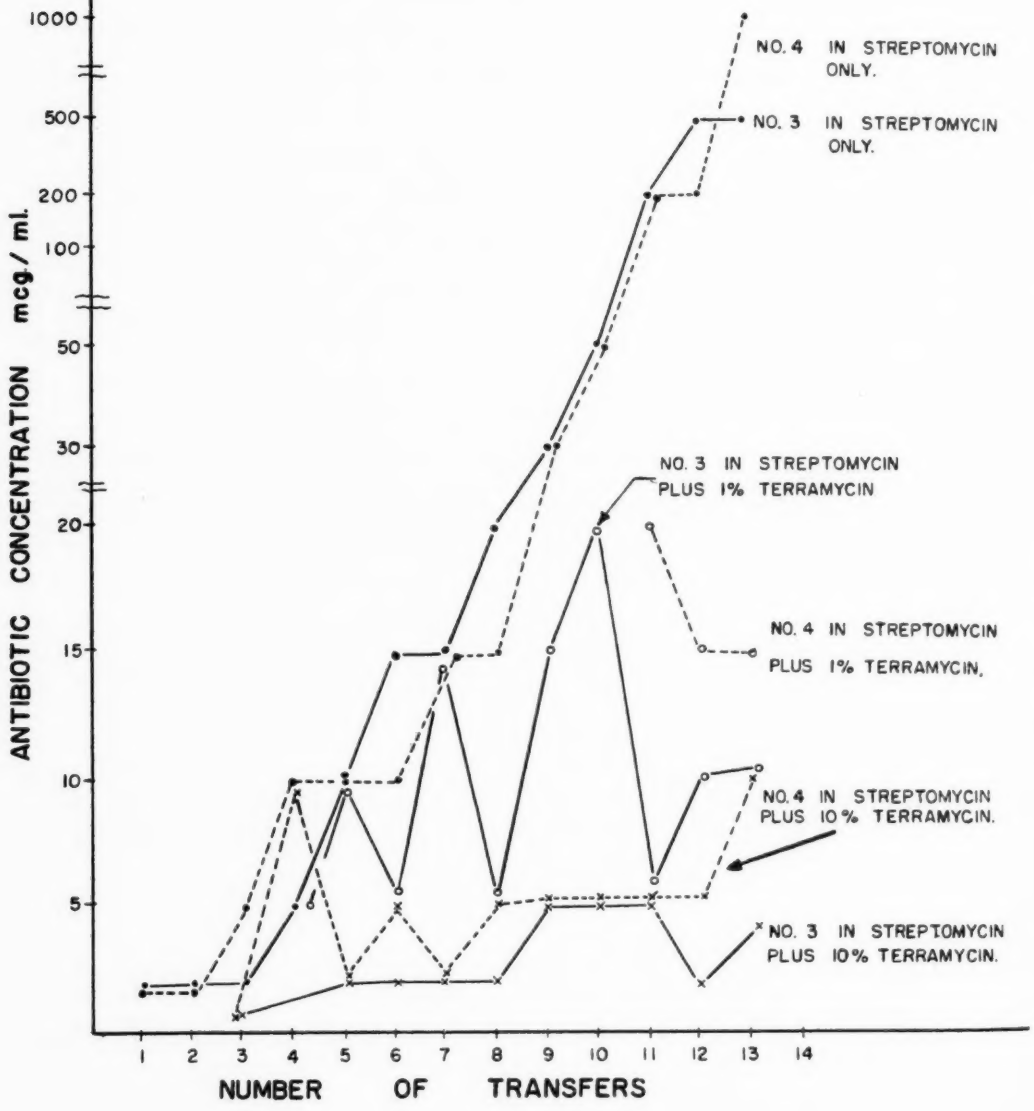
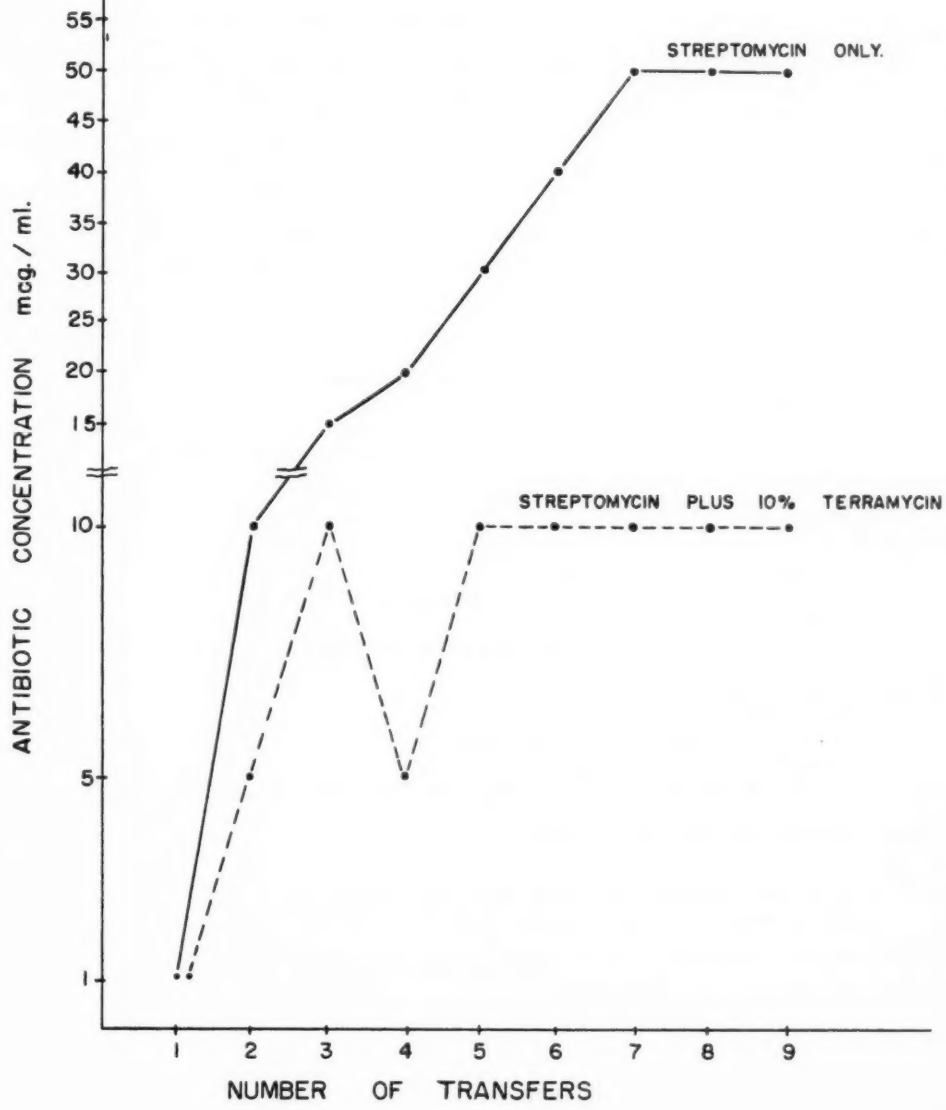


FIGURE 2

DEVELOPMENT OF RESISTANCE OF X. VESICATORIA TO STREPTOMYCIN, TERRAMYCIN, AND COMBINATIONS THERE OF.



BACTERIOLOGY LABORATORY OF CHAS. PFIZER AND CO., INC. BROOKLYN, NEW YORK

EFFECTIVENESS OF ANTIBIOTICS FOR APPLE FIRE BLIGHT CONTROL UNDER EPIDEMIC CONDITIONS IN PENNSYLVANIA

R. S. Kirby

In south-central Pennsylvania there has been the worst outbreak of fire blight (Erwinia amylovora) on apples and pears observed in many years. In some apple orchards near infected pear trees as much as one-half to two-thirds of the crop has been lost.

Fortunately, one of our antibiotics demonstrations was located in an apple orchard where fire blight was severe.

In a ten-year old orchard (mostly Opalescent and Gravenstein) in Franklin County a block of 115 trees was sprayed twice in bloom with Agrimycin normal strength at 9.34 oz. per 100 gallons (250 gallons covered 115 trees or 2.2 gallons per tree). A similar block adjoining the first was sprayed with pheno-lead in petal fall through second cover. The remainder of the orchard was sprayed with captan 2 lbs. in the regular schedule sprays. Agrimycin and pheno-lead sprayed areas received captan in all other regular sprays.

The first spray of Agrimycin was applied April 23, when 20 percent of the flowers of Opalescent were open and Gravenstein was in full bloom; at the second spray on April 27 Opalescent was in full bloom and all the petals had fallen from Gravenstein.

On June 3 when green apples were about 1 1/4 inches in diameter the number of blighted flower clusters per tree were determined. It was estimated that each tree had between 800 and 1000 flower clusters or points of possible infections. Results are as follows:

	Average number infections per tree
AGRIMYCIN	
Counts were made on 26 trees taken at random. The number of blighted flower clusters for each tree was:	
Opalescent -- 15 trees counted:	
0, 0, 0, 1, 0, 0, 0, 2, 1, 13, 0, 1, 0, 0, 0 -- total 18 blight infections . . .	1.2
Gravenstein -- 11 trees counted:	
0, 0, 0, 0, 0, 0, 1, 1, 2, 2, -- total 6 blight infections	.54
PHENO-LEAD	
Opalescent -- 6 trees counted:	
5, 3, 7, 70, 80, 6 -- total 171 blight infections	28.5
Gravenstein -- 5 trees counted:	
11, 20, 53, 202, 570 -- total 856 blight infections	171.2
REGULAR SPRAY SCHEDULE	
Opalescent -- 2 trees counted:	
25, 13 -- total 38 blight infections	19.
Gravenstein -- 6 trees counted:	
16, 166, 231, 25, 143, 233 -- total 814 fire blight infections	135

Table 1 summarizes the results obtained.

Table 1. Comparison of Agrimycin, pheno-lead, and regular spray schedule for control of fire blight on apple in a demonstration orchard in Pennsylvania.

Treatment	Average number of blight infections per tree		
	Opalescent	Gravenstein	Total
Agrimycin (2 bloom sprays)	1.2	.54	.92
Pheno-Lead	28.5	171.2	88.5
Regular Schedule	19.	135.	106.5

On the Agrimycin-sprayed trees blight infections usually failed to grow any appreciable distance down the spurs or into the branches. The growth of fire blight through the spurs and into the branches was pronounced in the regular and pheno-lead blocks.

PENNSYLVANIA STATE UNIVERSITY, STATE COLLEGE, PENNSYLVANIA

THIURAM POLYSULFIDES AS FIELD FUNGICIDES

Saul Rich and James G. Horsfall[1]

Our laboratory has long had an interest in the fungicidal effectiveness of organic sulfur compounds. In line with this continuing interest, we field tested, during 1953, a series of thiuram polysulfides sent to us by the Rohm and Haas Co., Philadelphia, Pa.. The composition and code numbers of these materials are as follows: a) Ethylenebisthiuram tetrasulfide (He 257); b) Tetramethylthiuram trisulfide (Lo 737); c) Ethylenebisthiuram trisulfide (Lo 738); and d) Tetramethylthiuram tetrasulfide (Lo 765). Fungitoxicity data for He 257 have been published by Barratt and Horsfall (1). The relative effectiveness of these materials for the control of bean powdery mildew in the laboratory and greenhouse has been reported by Keil, Froehlich, and Maughan (4).

In our field trials, the thiuram polysulfides, formulated as wettable powders, were applied by hydraulic equipment. These formulations were tested for their ability to control apple scab (Venturia inaequalis), early blight of tomatoes (Alternaria solani), and snapdragon rust (Puccinia antirrhini). The Horsfall-Barratt grading system (2) was used for estimating disease.

The apple scab trials were made on large McIntosh trees, using the half-tree technique (5). The sprays were applied at a pressure of 300 p. s. i. Spray dates were April 9, April 22, May 4, May 19, June 1, and June 16. Leaf scab data, taken on July 2, were based on reading 100 leaves per half tree or 400 leaves per treatment. Fruit set was insufficient for adequate fruit data.

Trials on tomato early blight and snapdragon rust were made in spirally arranged plots (3) using a spray pressure of 250 p. s. i., and a rate of 200 gallons per acre. The tomatoes (Chatham variety) were sprayed on August 26, and September 3, 9, and 16. The data, taken September 17, are based on estimated defoliation of 5 plants per plot, or 20 plants per treatment. The snapdragons (Afterglow variety) were sprayed on July 8, 15, 22, 31, August 6, 20, 27, September 4, 22, 30, and October 6. The plants were inoculated with rust on September 9, and the data were taken October 15. The data are based on estimated percentage of total leaf area involved by rust pustules for 5 plants per plot, or 20 plants per treatment. Standard fungicides were included as separate treatments in all trials.

The data (Table 1) are given in average percentage of disease as converted from average disease grade. The following discussion is based on analyses of variance of the disease grades. Statistical significance is expressed at the 5% level in all cases.

For controlling apple scab, all spray treatments were significantly better then the check. Based on effectiveness of control, the spray treatments could be separated statistically into three groups. The least effective group included both concentrations of He 257 and Lo 737, and the low concentrations of Lo 738 and Lo 765. The middle group included the high concentrations of Lo 738 and Lo 765, and the mixture of Fermate with sulfur. The most effective group included Phygon and Orthocide 406.

An analysis of the tomato early blight data showed that the following treatments were not significantly different from the check: Phygon, both concentrations of He 257 and Lo 737, and the low concentration of Lo 738. The following treatments were significantly better than the check: Dithane Z-78, Orthocide 406, the high concentration of Lo 738, and both concentrations of Lo 765. The treatments in the last group did not differ significantly one from the other.

All of the treatments for the control of snapdragon rust were significantly better than the check, except Phygon and the low concentrations of both He 257 and Lo 737. The following treatments, however, were less effective than Dithane Z-78 or the high concentration of He 257 for the control of snapdragon rust: Fermate, both concentrations of Lo 738 and Lo 765, and the high concentration of Lo 737.

The results of these field trials show that certain thiuram polysulfides are effective protectants against foliage diseases.

[1]The authors wish to acknowledge the helpful suggestions of Dr. W. E. Craig and Dr. C. P. Lo of the Rohm and Haas Co., and the able assistance of Mr. A. D. McDonnell.

Table 1. Comparative disease data for fungicide trials of thiuram polysulfides, from tests at Connecticut Agricultural Experiment Station, 1953.

Material	Concentration of formulation per 100 gal.	Average percent apple leaf area covered by scab	Average percent defoliation of tomatoes by early blight	Average percent snapdragon leaf area showing rust pustules
He 257	1/2	13.1	11.5	18.7
He 257	2	8.9	15.0[a]	3.3
Lo 737	1/2	12.0	11.5[a]	17.0
Lo 737	2	13.7	14.0[b]	11.7
Lo 738	1/2	11.8	9.0	8.0
Lo 738	2	6.6	6.5	5.4
Lo 765	1/2	7.3	4.5	14.0
Lo 765	2	6.0	4.0	12.5
Phygon XL	1/2	1.8	8.5	42.0
Orthocide 406	2	1.8	4.0	11.0
Fermate + Sulfur	1 + 3	3.0	---	---
Fermate	2	---	---	6.2
Dithane Z-78	2	---	3.0	3.0
Check	---	48.8	15.0	39.5

[a]Slight injury.
[b]Moderate injury.

Literature Cited

1. Barratt, R. W., and J. G. Horsfall. 1947. Fungicidal action of metallic alkyl bis-dithiocarbamates. Connecticut Agr. Exp. Sta. Bull. 508.
2. Horsfall, J. G. 1945. Fungicides and their action. 239 pp. Chronica Botanica Co., Waltham, Mass.
3. Horsfall, J. G., and Saul Rich. 1949. Spirally arranged plots in a design for field assay of fungicides. Connecticut Agr. Exp. Sta. Bull. 530.
4. Keil, H. L., H. P. Froehlich, and F. B. Maughan. 1953. Efficacy of certain organic compounds in control of bean powdery mildew under laboratory conditions. Phytopath. 43: 477.
5. Rich, S. 1952. Using half-tree plots for increasing the efficiency of fungicide tests. Phytopath. 42: 353-354.

DEPARTMENT OF PLANT PATHOLOGY, THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN 4, CONNECTICUT

DIPLODINA ROT OF SOUTH CAROLINA PEACHES

W. R. Wright, M. A. Smith, and G. B. Ramsey[1]

During July 1953 the Market Pathology laboratory in Chicago received five 1 1/9 bushel wire-bound test crates of Elberta peaches that had been included in a commercial shipment from South Carolina. These peaches were held for a week at room temperatures (80° to 90° F). During this period most of the fruit became infected with brown rot but a number in each

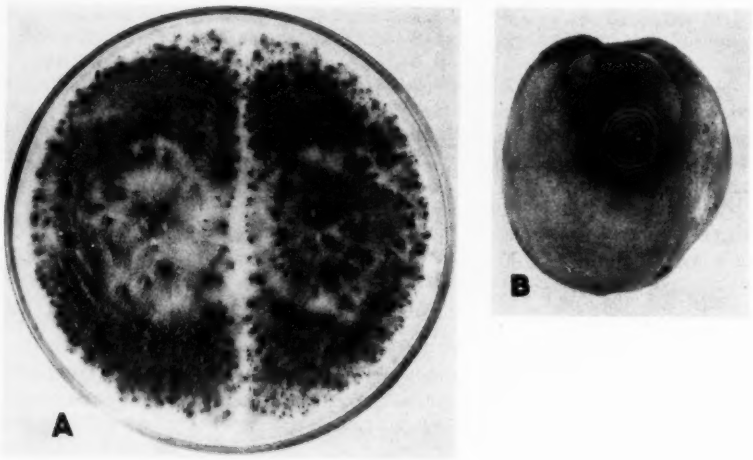


FIGURE 1. A -- Four-week-old culture of Diplodina persicae on white corn meal agar. B -- Natural infection of a South Carolina Elberta peach by D. persicae.

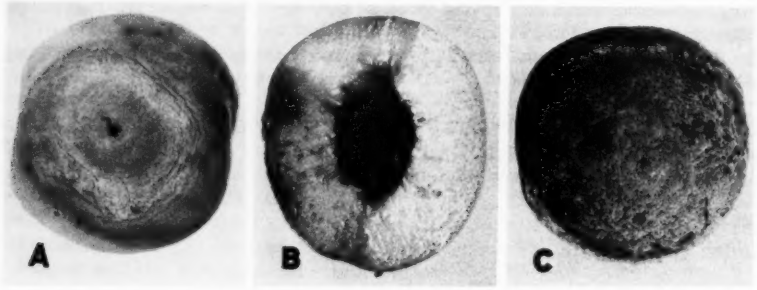


FIGURE 2. A -- Elberta peach six days after being inoculated with Diplodina persicae. B -- Cross section of peach shown in A. C -- Elberta peach inoculated with D. persicae, showing an early stage of mummification and abundant pycnidia.

[1]Respectively, Assistant Plant Pathologist, Plant Pathologist, and Principal Plant Pathologist, Biological Sciences Branch, Agricultural Marketing Service, U. S. Department of Agriculture. The work reported here was done in cooperation with the University of Chicago.

crate displayed infections not immediately identifiable. When zoning was conspicuous, the early symptoms of this infection appeared similar to anthracnose symptoms (Fig. 1, B). When zonation was not apparent the decay might have been confused with brown rot. As the rot progressed, the lesion was sharply delimited and a fine wrinkling of the darkening epidermal tissue occurred. Small black pycnidia averaging 180 microns, yielding hyaline, club-shaped to elliptic spores averaging 26 x 6.2 microns, were apparent at this stage. Spores were occasionally 1- to 3-septate, but the majority were continuous, a characteristic which did not appear to change with age in culture.

The fungus fruited well on potato-dextrose agar and on white corn meal agar. On the latter, growth was dark and appressed, and stromatic masses containing pycnidia (Fig. 1, A) were developed. Pycnospores averaged 22.8 x 7.2 microns.

Inoculations of market-ripe peaches by both water suspension of spores on sound fruit and insertion of inoculum in scalpel wounds produced lesions corresponding closely to the original infections. Progress of the decay was rapid, approximately half of a peach being affected in six days at 80° to 90° F (Fig. 2, A). Internal decay was equally rapid, the invaded tissue presenting a soft, moist, light-brown appearance (Fig. 2, B). Early stages of mummification were often evident a week after inoculation, with the affected area of the peach displaying slight sinking, wrinkling, and a leathery texture to the touch (Fig. 2, C).

Horn (1) and Horn and Hawthorne (2) described, from Louisiana, a peach disease which appeared to correspond closely to that caused by the organism herein described. Upon request, Dr. Horn kindly sent a culture of the fungus, which he tentatively identified as a Diplodina and which has recently been described as D. persicae Horn & Hawthorne. A comparison of the Louisiana and the South Carolina organisms did not reveal any significant differences.

The apparent ease with which the fungus infects the peach fruit, its demonstrated ability to spread by contact, and the rapidity with which it rots the fruit suggest a potential source of trouble to both grower and shipper. Temperature studies conducted by Horn and Hawthorne (2) indicate that little, if any, growth is made by this fungus at 55° F. It appears, therefore, that prompt precooling of peaches and shipment under standard refrigeration should effectively control development of decay by this fungus during transit.

Literature Cited

1. Horn, N. L. 1952. Diplodina on peaches in Louisiana. Plant Dis. Reptr. 36: 351.
2. Horn, Norman L., and Hawthorne, P. L. 1954. A new fruit rot of peach. Phytopath. 44: 134-136.

BIOLOGICAL SCIENCES BRANCH, AGRICULTURAL MARKETING SERVICE, U. S. DEPARTMENT OF AGRICULTURE

VERTICILLIUM WILT OF SWEET CHERRY IN CALIFORNIA

Stephen Wilhelm, George Nyland, and Fred Charles

During the 1952 and 1953 seasons symptoms suggestive of the Verticillium disease, and similar to those recently reported from British Columbia (6), were observed in sweet cherry varieties growing in the Davis, Santa Clara, and Stockton areas in California. Only occasional trees of considerable numbers were affected, and these were growing on the Prunus mahaleb and P. avium (Mazzard) understocks. Verticillium albo-atrum Reinke et Berth. was isolated from discolored wood of several of these trees and all isolates proved to be experimentally pathogenic to tomato (Pearson variety).

Outbreaks of Verticillium wilt have not been severe in the regions mentioned above, where recent cherry planting has been rather extensive, even though in some instances land with a tomato cropping history of several years was planted. Nothing like the serious outbreak recently reported by McIntosh (6) nor like the outbreaks commonly experienced in apricot or peach planted on land with a tomato or potato history, has yet been observed in cherry here.

Van der Lek of Holland (7) cited cherry (P. cerasus) as susceptible to the Verticillium disease in 1918. This appears to be the first report of the disease in cherry. His isolates of Verticillium from cherry were experimentally pathogenic to potato and cucumber. In 1925 Van der Meer (8) published a thorough study of the Verticillium disease of cherry in which she reported that both the common morello stock used in Holland (P. cerasus austera Ehrh.) and the rarely used P. mahaleb stock were susceptible to the disease. In Holland the disease was associated with the practice of inter-cropping young orchards with potatoes, and as here, the symptoms of yellowing and casting of older leaves and dying back of twigs, appeared suddenly in midsummer (July or August).

Reports, in addition to those from Holland and British Columbia (2, 6), indicate that the disease has been found in Czechoslovakia (1), France (4), Germany (3, 9)and possibly Italy (5). The report from Italy is not conclusive, since cultural evidence was not convincing and it is now known that symptoms such as wilting and die back may occur in trees affected with other diseases.

Literature Cited

1. Anonymous. 1926. Verticillosa třešní: (Verticilliosis of the cherry). Ochrana Rostlin 6: 32. 1926. (In Rev. Appl. Mycology 6: 238. 1927).
2. Conners, J. L., and D. B. O. Saville. 1945. New or noteworthy diseases. In 24th Ann. Rept. Canadian Plant Disease Survey. 1944. 122 p.
3. Donandt, S. 1932. Untersuchungen über die Pathogenität des Wirtelpilzes Verticillium albo-atrum R. u. B. Zeitschr. für Parasitenkunde 4: 653-711.
4. Dufrenoy, J., and M. L. Dufrenoy. 1927. Hadromycoses. Ann. des Épiphyties. 13: 195-212.
5. Lindegg, Giovanna. 1933. Un deperimento dei Ciliegi (a wilt of cherry trees). Riv. Pat. Veg. 23: 347-356. (In Rev. Appl. Myc. 13: 247. 1934).
6. McIntosh, D. L. 1954. Verticillium wilt of sweet cherry in British Columbia. Plant Dis. Reptr. 38: 74-75.
7. Van der Lek, H. A. A. 1918. Onderzoekingen over tracheomycosen: De Verticilliose van den Konkommer. Meded. Landbouwhoogesch., (Wageningen) 15: 1-36.
8. Van der Meer, Jikke H. H. 1925. Verticillium wilt of herbaceous and woody plants. Meded. Landbouwhoogesch., (Wageningen) 28: 1-82.
9. Wollenweber, H. W. 1929. Die Wirtelpilz-Welkekrankheit (Verticilliose) von Ulme, Ahorn und Linde usw. Arb. Biol. Reichsanst. für Land- und Forstwirtsch. 17: 273-299.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

TRISTEZA IN SATSUMA VARIETIES IN TEXAS[1]

Edward O. Olson and James R. McDonald

Satsuma oranges, a group of varieties belonging to Citrus reticulata, are the most important of the citrus fruits grown in northwestern Florida, southern Alabama, Mississippi and Louisiana, as well as in other sections too cold for round oranges and grapefruit. Some are also grown in California. Few satsumas are grown in the Lower Rio Grande Valley of Texas, but scattered plantings occur along the Gulf coast and in other sections of Texas.

Tristeza, or quick decline, a virus disease affecting orange, mandarin, and grapefruit trees on sour orange rootstock, has been found in California (4), Florida (9), and Louisiana (8). In Texas the Tristeza virus has recently been found in some Meyer lemon trees (15). This article reports the occurrence of Tristeza virus in 17 satsuma varieties and in Meyer lemon tops grown on trifoliate-orange (Poncirus trifoliata) or other rootstocks in a variety collection near Winter Haven, Texas.

METHODS AND RESULTS

Scionwood twigs were collected from trees of Meyer lemon, 26 varieties of satsumas, and several other citrus varieties growing near Winter Haven, Texas. The satsuma varieties, of local fame in Japan, were under test for possible superiority to the Owari, the standard variety in the United States (18). Two or more scion twigs from each tree were grafted, one twig each, to the main stem of Mexican lime seedlings growing in gallon cans of soil.

Within 60 to 120 days some of the grafted lime seedlings showed vein clearing, cupping and yellowing of the leaves and pitting of the stems similar to if not identical with the symptoms of tristeza on lime plants in Brazil (3), South Africa (11), California (19), and Florida (9). The symptoms were also similar to those previously obtained in Texas (15) from tests of tristeza transmission from Meyer lemons and indicate the presence of a mild strain of tristeza virus.

The results of these tests, presented in Table 1, show that a strain of Tristeza virus was present in trees of Meyer lemon and at least 17 of the 26 satsuma varieties growing in the citrus variety planting near Winter Haven, Texas.

DISCUSSION AND CONCLUSIONS

It has long been recognized that satsumas do poorly or fail entirely on sour orange rootstock, but grow well on sweet orange or trifoliate-orange rootstocks (10, 17, 20). In 1909 Swingle (17) considered satsumas on sour orange rootstock to be a complete failure, the growth being so slow and stunted that the satsumas never became more than dwarfed bushes; there was every evidence of a considerable degree of incompatibility between the satsuma and the sour orange. Swingle further noted: "Inquiry among growers of Satsuma oranges in Florida, Alabama, Mississippi, Louisiana, Texas and northern Mexico has failed to elicit a single instance of successful growth and satisfactory fruiting of this variety budded on sour-orange stock." In Florida (2) the sour orange is considered an unsatisfactory rootstock for satsumas. In California Webber (21) reported that satsumas on sour orange rootstock in his experiments began to die when 5 to 6 years old. Trees of Owari satsuma on Brazilian or Standard sour orange rootstocks were vigorous and produced good yields, while those on Rubidoux or African sour orange rootstocks were either nearly dead or in poor condition 10 years after they were planted in California (1). In this same experiment a similar failure of the Owari was noted on rootstocks of Duncan grapefruit and Coleman citrange, where no contributory disease could be found (22). In the Lower Rio Grande Valley of Texas, sour orange has been used almost exclusively as a rootstock, yet Friend and Wood (5) declared that Owari satsuma must be grown on citrange rootstock to be successful under Valley conditions.

However, there are reports of good growth and yields of satsumas on sour orange rootstock. In 1954 Mortensen (14) of Winter Haven, Texas, reported normal growth and good yields from Owari, Silverhill, and Kawano Wase trees on either sour orange or trifoliate orange rootstock. It seems significant that in the present tests of satsumas from the Winter Haven planting the Owari and Silverhill trees were found to be free of tristeza; the Kawano Wase was not tested.

[1]These investigations are a part of the Cooperative Citrus Rootstock Investigations conducted by the Texas Agricultural Experiment Station and the U. S. Department of Agriculture, certain phases of which were carried on under the Agricultural Marketing Act (R. M. A. Title II).

Table 1. Occurrence of Tristeza virus in trees of satsumas and other citrus varieties on various rootstocks, in a variety planting at Winter Haven, Texas

Group and variety or type of top	Rootstock variety or type	Year planted
Group affected with Tristeza virus[a]:		
Bito Wase satsuma	Citrumelo[b]	1938
Foley 11481 satsuma	Rusk citrange[c]	1932
Foley 11554 satsuma	Trifoliate-orange	1934
Higuchi satsuma	Cunningham citrange	1933
Ikiriki satsuma	Savage citrange	1933
Iwakuma satsuma	Rusk citrange	1938
Matsui satsuma	Cunningham citrange	1938
Miyasaki satsuma	Carrizo citrange	1941
Natsume satsuma	Grapefruit	1938
Oba satsuma	Rusk citrange	1938
Omura satsuma	Savage citrange	1933
Osuga satsuma	Citrumelo	1938
Saruwatari satsuma	Cunningham citrange	1932
Shintani satsuma	d	1946
Sueoka satsuma	Trifoliate-orange	1932
Suzuki satsuma	Rustic citrange	1938
Yakushiji satsuma	Savage citrange	1934
Meyer lemon	Trifoliate-orange	1931
Meyer lemon	d	1932
Group free of Tristeza virus[e]:		
Aikawa satsuma	Trifoliate-orange	1936
False Hybrid satsuma	Trifoliate-orange	1933
Horie satsuma	Morton citrange	1932
Hozaki satsuma	Trifoliate-orange	1932
Morita satsuma	Cunningham citrange	1933
Owari satsuma	Trifoliate-orange	1931
Silverhill satsuma	Trifoliate-orange	1932
Yamazaki satsuma	Trifoliate-orange	1935
Yanosaki satsuma	Trifoliate-orange	1935
Little River grapefruit	Carrizo citrange	1939
Marsh Pink grapefruit	Citrumelo	1938
Joppa orange	Trifoliate-orange	1934
Purvis orange	Citrumelo	1937
Wekiwa tangelo	Cunningham citrange	1939
Navel orange	Sweet orange	1943
Nagami kumquat	Trifoliate-orange	1931

[a]Determined by symptoms of tristeza (vein clearing and stem pitting) on Mexican type lime seedlings following scionwood graft inoculations.
[b]Citrumelo is a hybrid (trifoliate-orange x grapefruit).
[c]Citranges are hybrids (trifoliate-orange x sweet orange).
[d]Own-rooted.
[e]No symptoms in 120 days on lime test plants.

The history of poor growth of satsumas on tristeza-intolerant sour orange rootstock and good growth on tristeza-tolerant trifoliate-orange and sweet orange rootstocks, coupled with the findings in the present tests of Tristeza virus in 17 of 26 satsuma selections, suggests that Tristeza virus may have been present for many years in many of the satsuma varieties grown in various parts of the United States. The fact that satsuma varieties are propagated by budding may account for the widespread virus distribution, although some distribution may have occurred through aphid vectors of the virus.

The trifoliate-orange, the Cunningham, Morton, Rusk, and Savage citranges, and the Winter Haven citrumelo were among the rootstocks tolerant to the strain of Tristeza virus

studied in Brazil (6). A later report from Brazil (7) noted differing degrees of tristeza symptoms and stem pitting on these trifoliate-hybrid rootstocks. The results from Winter Haven (Table 1) confirm the finding (6) that some citranges are tristeza-tolerant rootstocks, since satsuma trees more than 20 years old still lived and bore fruit in the presence of Tristeza virus. However, satsumas at Winter Haven have shown leaf bronzing the past few years, even on trees free of Tristeza virus, which may have obscured the differences in tristeza tolerance among rootstocks.

The determination of the presence of Tristeza virus in trees representing 17 satsuma varieties in the Winter Haven area adds one more disease to the lengthening list of probable causes of the previously reported (12, 13) "Winter Haven decline". The "Winter Haven decline" has been compared to "spreading decline" and "blight" in Florida (12) and has been considered to be related to stress caused by tree crowding on a shallow soil and included exocortis infection of trifoliate-orange rootstocks (13, 14). Pythium ultimum was considered a probable contributing cause of "Winter Haven decline" (16). Since some orange trees showing either exocortis on the rootstock or "Winter Haven decline" of the top have been found to be free of Tristeza virus, it seems that tristeza is not the only cause of the "Winter Haven decline".

Literature Cited

1. Batchelor, Leon D., and Marvin B. Rounds. 1948. Choice of rootstocks. Chap. 3, Vol. 2, The Citrus Industry. Univ. of California Press. pp. 169-222.
2. Camp, A. F. 1931. Citrus propagation. Florida Agr. Expt. Sta. Bul. 227: 1-48.
3. Costa, A. S., T. J. Grant, and S. Moreira. 1950. On a possible relation between tristeza and the stem pitting disease of grapefruit in Africa. California Citrograph 35: 526-528.
4. Fawcett, H. S., and J. M. Wallace. 1946. Evidence of the virus nature of citrus quick decline. California Citrograph 32: 50, 88, 89.
5. Friend, W. H., and J. F. Wood. 1941. Citrus varieties for the Lower Rio Grande Valley. Texas Agr. Expt. Sta. Bul. 601: 1-36.
6. Grant, T. J., A. S. Costa and S. Moreira. 1949. Studies of tristeza disease of citrus in Brazil. Florida State Hort. Soc. Proc. 62: 72-79.
7. _____, _____ and _____. 1951. Tristeza disease of citrus in Brazil. California Citrograph 36 (8): 310, 311, 324-326, 328, 329.
8. _____, L. J. Klotz, and J. M. Wallace. 1953. The tristeza disease of citrus. Plant Diseases, the Yearbook of Agriculture 1953. U. S. Department of Agriculture. pp. 730-734.
9. _____, and Henry Schneider. 1953. Initial evidence of the presence of tristeza, or quick decline, of citrus in Florida. Phytopath. 43: 51-52.
10. Hume, H. Harold. 1909. Citrus fruits in Texas. Texas Dept. Agr. Bul. (New series) 3: 1-33.
11. McClean, A. P. D. 1950. Virus infections of citrus in South Africa. Farming in South Africa 25 (293): 262; 25 (294): 289.
12. Mortensen, E. 1947. Citrus tree decline in the Winter Garden area. Proc. Lower Rio Grande Valley Citrus & Veg. Inst. 2: 84-87.
13. _____. 1952. Citrus tree decline at Winter Haven, Texas. Proc. Rio Grande Valley Hort. Inst. 6: 45-47.
14. _____. 1954. Citrus rootstocks in the Winter Garden area of Texas. Proc. Rio Grande Valley Hort. Inst. 8: 13-22.
15. Olson, Edward O. and Bailey Sleeth. 1954. Tristeza virus carried by some Meyer lemon trees in South Texas. Proc. Rio Grande Valley Hort. Inst. 8: 84-88.
16. Sleeth, Bailey. 1953. Winter Haven decline of citrus. Plant Dis. Reptr. 37: 425-426.
17. Swingle, Walter T. 1909. The limitation of the satsuma orange to trifoliate-orange stock. U. S. Dept. Agr. Bur. Plant Industry Circ. 46: 1-10.
18. Traub, Hamilton P., and T. Ralph Robinson. 1937. Improvement of subtropical fruit crops: citrus. Yearbook of Agriculture 1937. U. S. Department of Agriculture. pp. 749-826.
19. Wallace, J. M. and R. J. Drake. 1951. Newly discovered symptoms of quick decline and related diseases. Citrus Leaves 31: 8-9, 30.
20. Webber, Herbert J. 1897. Methods of propagating the orange and other citrus fruits. U. S. Department of Agriculture Yearbook, 1896. pp. 471-489.

21. Webber, Herbert J. 1935. Rootstocks for the satsuma orange. Pacific Rural Press 129 (March): 334, 338.
22. _____. 1948. Rootstocks: their character and reactions. Chap. 2, Vol. 2, The Citrus Industry. Univ. of California Press. pp. 69-168.

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, AND TEXAS AGRICULTURAL EXPERIMENT STATION, WESLACO, TEXAS

A DISEASE OF PECANS RESEMBLING BOTH ROSETTE AND BUNCH COLLECTED IN NEW MEXICO

John R. Cole

Specimens of pecan branches with characteristics of both the virus disease, bunch, and the deficiency disease, rosette, were recently received from Las Cruces, New Mexico, in the Mesilla Valley. Las Cruces is approximately 60 miles west of El Paso, Texas. These specimens were collected by Lieutenant Colonel Samuel O. Hill[1], Medical Entomologist, located at Brooke Army Medical Center, Fort Sam Houston, Texas.

FIGURE 1. Pecan tree growing on the White Sands Proving Ground near Las Cruces, New Mexico, illustrating characteristics of both rosette and bunch disease. Photograph courtesy of the U. S. Army.

[1]Colonel Hill was Entomologist in charge of the Pecan Insect Laboratory, Monticello, Florida, before he entered the military service during World War II.

The characteristic symptoms of the disease from Las Cruces are a bushy or broomy growth of slender, willowy shoots resulting from abnormal forcing into growth of lateral buds (Figure 1). This multiplicity of branching gives the diseased portions of the tree a witches'-broom or "bunching" effect. These symptoms are typical of bunch disease. However, the leaves on the specimens submitted by Colonel Hill are mottled or chlorotic, crinkled and narrow, symptoms which are characteristic of rosette. These contrast with symptoms of bunch; on trees affected with bunch the diseased leaves vary from dark green to chlorotic, but the terminal leaves are subnormal in length and usually all the leaflets are abnormally thin and of more than normal breadth. Furthermore, the leaflets on shoots affected with bunch are frequently wavy or twisted and give an appearance of being slightly wilted.

According to Colonel Hill the soil where the pecan trees are growing at Las Cruces has a pH of 8.5. The writer has observed trees under cultivation rosetting severely at a pH of this level.

So far as the writer has been able to ascertain this is the first time that this malady has been reported from New Mexico. Trees known to be affected with bunch disease have been observed in Mississippi, Louisiana, Arkansas, Texas, and Oklahoma, while rosette is prevalent in most areas where pecans are grown unless the trees are treated with zinc sulfate to prevent or overcome the disease.

Colonel Hill did not state the variety affected, but he did say that the trees are budded, growing under irrigation, and located on a military reservation. The Schley and Mahan are among the varieties most susceptible to bunch disease, while the Stuart variety is highly resistant. All varieties are affected by rosette, but the Stuart is the most susceptible and the Moneymaker the most resistant.

U. S. PECAN FIELD STATION, HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, ALBANY, GEORGIA

TRANSMISSION OF WHEAT STREAK-MOSAIC VIRUS BY ABRASIVE LEAF CONTACTS DURING STRONG WINDS[1]

W. H. Sill, Jr., Alvin E. Lowe, R. C. Bellingham, and H. Fellows[2]

In 1953, Slykhuis (7) reported that the eriophyid mite, Aceria tulipae (K.), was a vector of the Wheat Streak-Mosaic virus. This was confirmed by Staples and Connin (8) and there is now no doubt that this mite is one of the principal vectors. From 1949 to 1952 various workers in Kansas, South Dakota, and Nebraska had failed to find a vector of this disease or to confirm the report of Atkinson (1) that the greenbug, Toxoptera graminum (Rond.) transmitted it. Other suggested means of transmission were grazing and trampling of livestock, moving of machinery, strong winds, and blowing soil. These were all investigated. Wind causes an intermingling and lashing of leaves between adjacent diseased and healthy plants and might abrade the leaves enough to inoculate some healthy plants. Although this virus is not soil-borne (3, 6), blowing soil particles striking diseased plants might pick up infective aliquots of the virus and then inoculate healthy plants upon impact.

Materials and Methods

Experiments to determine the importance of livestock grazing and trampling and of moving machinery in the transmission of this virus in Reward spring wheat were conducted at the Garden City and Hays, Kansas, branch experiment stations, the spring of 1952. Three other tests were made with Comanche winter wheat during the 1952-53 season. In each experiment, 16 rows, 120 feet long, were sown and six to eight of these rows were inoculated with the Wheat Streak-Mosaic virus, using the spraying technique developed by McKinney and Fellows (4). The whole plot was divided into four 30-foot blocks each containing inoculated and uninoculated parts. After the appearance of definite symptoms in the inoculated rows, one block was grazed heavily by cattle, alternately on diseased and healthy parts of the block. In another block the cattle were led alternately over diseased and healthy areas, but were not permitted to graze. A light Ford tractor was driven 25 round trips over diseased and healthy areas in the third block. The fourth block was left for a control.

For the wind and blowing soil experiments, a wind tunnel which had been developed by the High Plains Wind Erosion Laboratory now located at Kansas State College, and described by Zingg and Chepil (10), furnished strong winds approximating 30 m.p.h., 6 inches above the soil surface. This was considerably in excess of average winds typical of the present Wheat Streak-Mosaic region (9).

Silt loam soil similar to that which blows over much of western Kansas was collected near Phillipsburg. It was screened to 0.59 mm. particle size and was used in the wind tunnel whenever blowing soil was desired. When soil artificially infested with the virus was desired, 2 quarts of a 1 to 20 (plant sap-water) dilution of the Wheat Streak-Mosaic virus was sprayed with a paint sprayer on each 250 lbs. of the screened silt loam. Applying more than this amount tended to retard the blowing of this soil. The infested soil was used in the wind tunnel on the day that it was prepared. The soil particles doubtless were carrying much more virus inoculum than could have been picked up from infected plants in the field.

Westar wheat plants were grown either in greenhouse flats or in 6-inch clay pots in a well fumigated greenhouse at temperatures between 70° to 80° F. Some plants were inoculated at the three-leaf stage by the carborundum leaf rubbing method with a 1 to 5 dilution of the virus.

---

[1]Contribution No. 558, serial No. 437, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan, and contribution number 4, Garden City Branch Agricultural Experiment Station, Garden City, Kansas, and the Field Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture, cooperating.

[2]Assistant Professor of Botany, Kansas State College, Associate Agronomist, Garden City Branch of Kansas Experiment Station, Agent and Pathologist, Field Crops Research Branch, U. S. Department of Agriculture, respectively.

The authors wish to thank Mr. A. W. Zingg, Senior Agr. Engin., U.S.D.A., and Dr. W. S. Chepil, Professor of Agronomy, Kansas State College, for permission to use the wind tunnel of the High Plains Wind Erosion Laboratory located at Kansas State College, Manhattan, Kansas, and for technical assistance given during some experiments.

In other cases one-half of the plants in each 6-inch clay pot were inoculated. Alternate rows of wheat in greenhouse flats also were inoculated. Diseased and healthy plants were thus close together but transmission between diseased and healthy plants via root contacts is unlikely as reported by Fellows et al. (2). Other pots and flats were left uninoculated. The diseased and healthy plants were placed in the wind tunnel in various combinations when about one month of age (usually two to three tiller stage). Each experiment was triplicated using an approximate total of 300 plants per experiment.

Experimental Results

In the livestock grazing and trampling and moving machinery tests, very few plants in the uninoculated areas developed mosaic symptoms. Manual inoculations from suspected to healthy plants were made by the carborundum rubbing method as a further check. A few plants contained the virus but as many of these came from the check plots as from any of the three treatments, indicating that the three treatments were not involved in transmission. However, in one experiment at Hays in the spring of 1953, the uninoculated areas of both the control block and the three treatment blocks were 50 to 100 percent infected with wheat streak mosaic. Later, when the report of Slykhuis (7) was seen, these blocks were examined and a large population of the mite A. tulipae was found on the wheat. Mites were not found at the Garden City plots and transmission results there were negative.

In another series of experiments sheep were fed alternately on pots of diseased and healthy wheat. No transmission to the healthy plants occurred in several trials.

In the wind and blowing soil experiments, tests first were made to determine the capacity of greenhouse grown plants to survive the effects of wind alone and of wind plus blowing soil. In three trials, one-month-old plants were exposed for 3, 6, 9, 12, and 20-minute periods. With wind alone there was no appreciable killing of plants even at 20 minutes, but visible leaf injury increased with blowing time. Mechanical injury to the plants was increased greatly by the addition of soil in the wind tunnel. Injury was slight at 6-minute exposures, medium to severe at 10 minutes, severe and occasionally lethal at 12 minutes, and usually lethal at 20 minutes. Consequently, the exposure time of most of the wind-tunnel tests where the wind-soil combination was used was reduced to 12 minutes or less. Where wind alone was employed, 20-minute exposures were general. No healthy plants developed the disease when wind or wind and inoculated or uninoculated soil was blown through them.

When pots or flats were placed in the tunnel so that wind could blow through adjacent, intermingling diseased and healthy plants, a total of 7.8 percent of the healthy plants developed the disease. When soil was blown through adjacent, intermingling diseased and healthy plants, 5.5 percent of the healthy plants became infected. The blowing soil increased leaf damage but slightly reduced disease transmission. The soil tended to accumulate around the exposed plants and gradually decreased the amount of leaf movement and possibly transmission also.

When artificially infested soil was blown through adjacent, intermingling diseased and healthy plants, only 2 percent of the healthy plants became diseased.

Leaves of diseased and healthy plants were rubbed alternately with the fingers without an abrasive causing some injury comparable to that caused by the lashing of leaves in the wind. Ten percent of all rubbed healthy plants developed the disease, whereas untreated healthy plants remained disease free. McKinney and Fellows (4) infected a low percentage of healthy plants by a similar technique.

Three experiments were devised to check the effect of proximity of diseased and healthy plants. In the first test, half of the plants in each 6-inch pot were inoculated, which allowed diseased and healthy plants to grow close together. After exposure of these pots to wind, 15 percent of all healthy plants became infected. In the second test, alternate rows of plants in greenhouse flats were inoculated and 14 percent of all uninoculated plants developed the disease after exposure to the wind. In the third test, separate pots of diseased and healthy plants were set adjacent to each other in alternate rows in the wind tunnel. Some of the diseased and healthy plant leaves were too far apart to interlace as the wind blew and only 6 percent of all healthy plants developed the disease. When flats of diseased plants were set adjacent to flats of healthy plants in the tunnel so that only outer rows of diseased and healthy plants could intermingle to any degree, only 5.5 percent of the healthy plants developed the disease after blowing.

When plants large enough for some diseased and healthy leaves to intermingle, were placed in the tunnel, 4.5 percent of the healthy plants developed the disease, but when the plants used were too small for the leaves to intermingle, no disease developed among the healthy

plants.

Discussion and Summary

A small amount of transmission of the Wheat Streak-Mosaic virus may occur during strong winds because of direct abrasive leaf contact between diseased and healthy wheat plants. Soil particles in the wind do not increase the percentage of transmission.

Transmission by direct abrasive leaf contact may be less under field conditions than in the wind tunnel experiments reported here because the winter wheat plants in the field tend to be prostrate in the fall and early spring when most of the blowing occurs. This would minimize leaf contact between diseased and healthy plants. Virus infection must happen early in the life of a wheat plant for severe damage to occur (5), which in Kansas means that early fall infection must occur in most years. Leaf contact between small plants is limited. In addition, in Kansas, winds are typically less prevalent and severe in the fall than in the spring (9). Wind in the field, as in the wind tunnel used in these experiments, causes blowing soil to accumulate around the young plants, thus retarding leaf to leaf abrasive contact somewhat.

All data obtained indicate that grazing and trampling of livestock and moving of machinery are not involved in the transmission of this virus disease in the field. The evidence obtained supports the contention of Slykhuis (7) that the eriophyid mite, Aceria tulipae (K.), is an important vector.

Literature Cited

1. Atkinson, R. E. 1949. Western wheat mosaic in Colorado and its transmission by the grain aphid, Toxoptera gramineum. (abstr.) Phytopath. 39: 2.
2. Fellows, H., W. H. Sill, Jr., and H. H. McKinney. 1952. Relative abundance of wheat yellow streak-mosaic virus in leaves, crowns, and roots of the wheat plant. Plant Dis. Reptr. 36: 319-320.
3. McKinney, H. H. 1937. Mosaic diseases of wheat and related cereals. U. S. Dept. Agr. Circ. No. 442.
4. McKinney, H. H., and H. Fellows. 1951. A method for inoculating varietal test nurseries with wheat streak-mosaic virus. Plant Dis. Reptr. 35: 264-266.
5. Sill, W. H., Jr. 1953. Some characteristics of the wheat streak-mosaic virus and disease. Trans. Kansas Acad. Sci. 56: 418-424.
6. Slykhuis, J. T. 1952. Virus diseases of cereal crops in South Dakota. South Dakota A. & M. College Tech. Bul. 11.
7. Slykhuis, J. T. 1953. Wheat streak mosaic in Alberta and factors related to its spread. Canad. Jour. Agric. Sci. 33: 195-197.
8. Staples, Robert, and R. V. Connin. --- Univ. of Nebraska and Kansas State College. (manuscript in preparation).
9. Zingg, Austin W. 1949. A study of the movement of surface wind. Agr. Eng. 30: 11-13.
10. Zingg, A. W., and W. S. Chepil. 1950. Aerodynamics of wind erosion. Agr. Eng. 31: 279-282.

KANSAS AGRICULTURAL EXPERIMENT STATION AND THE FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE

SOME HOSTS OF FEATHERY MOTTLE VIRUS OF SWEETPOTATO

Raymon E. Webb[1]

Summary

Plants of 33 varieties and species of families Convolvulaceae, Solanaceae, Leguminosae, and Cucurbitaceae were mechanically inoculated with the Feathery Mottle virus. Plants of the six varieties of sweetpotato (Ipomoea batatas (L.) Lam.), and of Calonyction aculeatum (L.) House, Quamoclit lobata (Cerv.) House, Ipomoea purpurea (L.) Roth var. Crimson Rambler, and Merremia sibirica (Pers.) Hall. f., were found susceptible to infection. Symptoms induced on the first three hosts were similar. Initial symptoms expressed by M. sibirica and Crimson Rambler morning-glory resembled those on the other three hosts; however, the small chlorotic dots in the leaf lamina were closely followed by extensive veinal necrosis over the infected leaves. Secondary symptoms on these latter two hosts were similar to the late primary symptoms. These distinctive symptoms make Crimson Rambler a good indicator plant for the Feathery Mottle virus.

Sweetpotatoes are among the most important horticultural crops grown in the South. The success of the sweetpotato industry depends largely upon rigid regulations governing the production and sale of certified seed (seed roots) and young sprouts. Millions of young sprouts produced from certified seed are needed each year to plant the commercial crop. To maintain this supply of certified seed and sprouts some States have established foundation-seed farms, under the direction of qualified personnel, to produce small quantities of hill-unit-selected, high-quality roots of the commonly grown and newer varieties. The output from these seed farms insures certified-seed growers a continual source of sweetpotato seed relatively free of fungal and virus diseases.

Although virus or virus-like diseases of the sweetpotato have been reported in the United States since 1920 (5, 6, 10, 11), they have not constituted a serious problem until recently. Since 1944 (3, 4) the Internal Cork virus has presented a menace through the sale of infected roots; however, little effect of it on root production has been noted. Borders and Ratcliff (1) have described the effects of what appears to be an insect-transmitted virus posing a potential threat to sweetpotato production in Georgia. Sheffield (7) has proven the virus nature of some sweetpotato diseases that are limiting factors to production in certain areas of Africa. In 1945 Doolittle and Harter (2) reported the Feathery Mottle virus of sweetpotato but stated that it was apparently not common in the field. Webb and Larson (8) found a mechanically transmissible virus in sweetpotato plants growing in Wisconsin and Louisiana. Comparative studies of the virus and the Feathery Mottle virus indicated that they are very similar if not identical. Aphid-transmission studies of the Feathery Mottle virus indicated that it could be spread from plant to plant by Myzus persicae (Sulz.) under greenhouse conditions.

Since field spread of the Feathery Mottle virus was shown possible, host-range studies were initiated to determine the role of naturally-occurring weeds and cultivated members of the family Convolvulaceae in harboring this virus. Many of these perennial weeds and cultivated plants are found growing widespread over the potato-growing areas. If susceptible to the virus, these plants could present a serious problem to production and maintenance of virus-free seed stocks.

Under field conditions, particularly during the bright, hot summer days, distinctive symptoms of the Feathery Mottle virus in varieties Porto Rico Unit 1, Georgia Bunch, and Texas Bunch are partially masked. The small chlorotic dots and subsequent vein-feathering symptoms on the young leaves fade rapidly, giving the entire plant a slightly chlorotic appearance. Only in the shaded portions of the interior of infected plants can definite symptoms be distinguished.

In root-transmission studies Webb and Larson (8) found that a period of 39 to 45 days was required for plants to show symptoms when grown from infected roots. This long incubation period makes it difficult,to rogue diseased plants and also makes the sweetpotato plants an unsatisfactory host for the virus-transmission studies. A true-seed host adapted to greenhouse

---

[1] Plant Pathologist, Horticultural Crops Research Branch, Agricultural Research Service, United States Department of Agriculture.

culture which would react distinctively to infection in a relatively short time was needed. A pathologist could use such a host to index foundation-and certified-seed stocks, detect the virus in suspected plants, and learn the properties of the virus. In this study on the possible suitability of different host plants, members of the families Solanaceae, Leguminosae, and Cucurbitaceae were included in addition to varieties and species of the family Convolvulaceae. A preliminary report of this study has been presented (9).

Materials and Methods

Seed of the various plants to be tested were planted in vermiculite. When the seedlings were in the second-leaf stage, they were transplanted to soil in 3-inch pots. Mechanical inoculations were made as soon as the plants recovered from the shock of transplanting.

Feathery-Mottle-virus-infected sweetpotato plants[2] served as the source of inoculum unless otherwise stated. Inoculum was applied with sterile cheesecloth and carborundum was used as an abrasive. After inoculation plants were sprayed with an insecticide twice weekly until discarded.

Results of Host Range Studies

Plants of the varieties and species to be inoculated were arranged in groups of ten each. Five plants of each were inoculated with expressed sap diluted 1-10. The other five plants of each series served as controls. The varieties and species inoculated, number of plants infected, and the incubation period are shown in Table 1. Only one series of inoculations was made on several species of plants because of a shortage of seed. Where possible, three to five separate series of inoculations were made to verify susceptibility or resistance to infection with the virus.

Of a total of 33 different varieties and species tested, plants representing only four genera, all of the family Convolvulaceae, were found susceptible to infection by the Feathery Mottle virus. The six varieties of Ipomoea batatas were readily infected and showed typical feathery mottle symptoms (Fig. 1). Symptoms expressed by infected plants of Calonyction aculeatum were similar to those shown by infected sweetpotato plants. Under greenhouse conditions vein feathering failed to develop as rapidly in these plants as in sweetpotato plants and the progress of the virus into the developing leaves was much slower. Infected plants were severely stunted, but necrosis of stems and leaves was not observed. Plants of Merremia sibirica which were infected developed a chlorotic band along the leaf veins. As the leaves matured, they became slightly chlorotic and the plants were severely dwarfed. Symptoms produced on infected plants of I. purpurea variety Crimson Rambler and on Quamoclit lobata were very similar. On the inoculated leaves, typical small chlorotic spots characteristic of infection by the Feathery Mottle virus developed (Fig. 2, B and C) slight vein clearing and vein necrosis of many small veinlets. Necrosis extended into the leaf lamina a short distance, and, as the leaves aged, portions of the necrotic areas dropped out giving the leaves a tattered and slightly chlorotic appearance (Fig. 2 D). The more severely affected leaves eventually abscised. Systemic symptoms were similar to those produced on the parts initially inoculated. As the infected plants grew, the young leaves, one-third to one-half grown, developed a slight mottle and vein clearing and veinal necrosis followed closely. Infected plants of both species failed to show severe stunting.

The presence or absence of the virus in inoculated plants of the various varieties and species was verified by subinoculations to sweetpotato and Crimson Rambler morning-glory plants. Subtransfers of the virus from infected Crimson Rambler and from the original virus source plants to sweetpotato and Crimson Rambler plants proved that the symptoms induced on Crimson Rambler were caused by the Feathery Mottle virus.

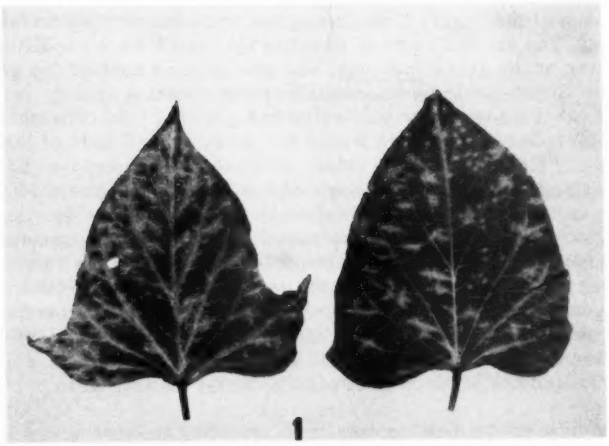
Virus-infected plants of Crimson Rambler blossomed and set seed freely. Seed from four infected plants were harvested and planted to determine whether the virus was seed-transmitted. All seeds were set after the plant was infected by the virus. Duplicate plantings were made with 200 seeds from each infected plant. The seedlings were observed for a 30-day period and all plants remained healthy.

---

[2]Supplied by Dr. S. P. Doolittle, Plant Industry Station, Beltsville, Maryland.

Table 1. Species and varieties of Convolvulaceae, Solanaceae, Leguminosae, and Cucurbitacae tested for susceptibility to the Feathery Mottle virus.

FAMILY Species Variety	Number of Plants Inoculated	 Infected	Incubation period (days)
CONVOLVULACEAE			
Calonyction aculeatum (L.) House	10	6	15-19
Ipomoea spp., horticultural varieties			
Blue Star	15	0	
Cornell	25	0	
Cyprus Vine	25	0	
Darling	25	0	
Pearly Gates	15	0	
Pure White	25	0	
Summer Skies	25	0	
I. batatas (L.) Lam.			
Heart O'Gold	20	9	14-18
Georgia Bunch	45	37	14-18
Porto Rico 2-1	20	14	14-18
Porto Rico Unit 1	45	32	14-18
Ranger	20	11	14-18
Texas Bunch	45	36	14-18
I. cairica (L.) Sweet	5	0	
I. nil (L.) Roth			
Scarlet O'Hara	25	0	
I. rubra (Vahl) Millsp.	15	0	
I. sinuata Ort. (I. dissecta)	5	0	
I. tricolor Cav.			
Heavenly Blue	25	0	
I. purpurea (L.) Roth			
Crimson Rambler	45	39	14-18
Merremia sibirica (Pers.) Hall. f.	5	2	15-17
Quamoclit coccinea Moench	5	0	
Q. lobata (Cerv.) House	5	2	15-17
Q. sloteri Nieuwl.			
Cardinal Climber	25	0	
SOLANACEAE			
Physalis floridana Rybd.	10	0	
Datura stramonium L. var. tatula (L.) Torr.	10	0	
Nicandra physalodes (L.) Gaertn.	10	0	
Nicotiana tabacum L. Connecticut Havana 38	13	0	
N. glutinosa L.	10	0	
N. rustica L.	10	0	
N. sylvestris Speg. & Comes	10	0	
LEGUMINOSAE			
Vigna sinensis (Torner) Savi			
Black	22	0	
CUCURBITACEAE			
Cucumis sativus L.			
National Pickling	23	0	



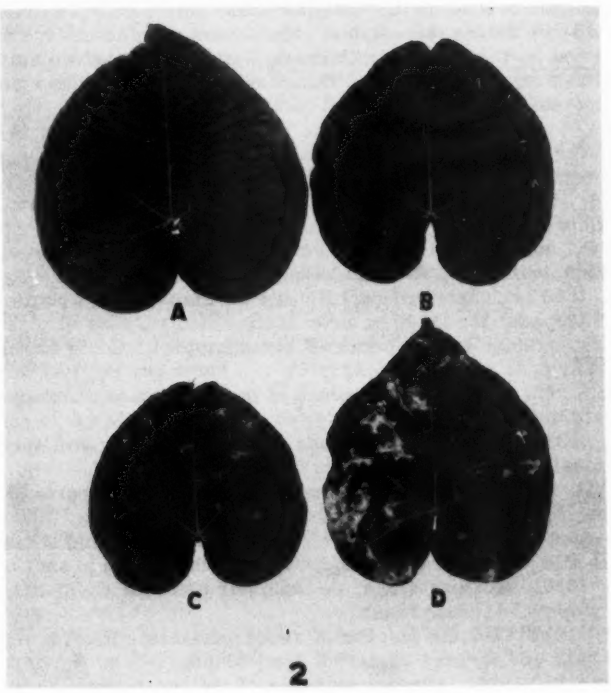


FIGURE 1. Leaves of Porto Rico Unit 1 sweetpotato showing symptoms of the Feathery Mottle virus.

FIGURE 2. A, Healthy leaf of Ipomoea purpurea variety Crimson Rambler; B, initial symptoms of infection by the Feathery Mottle virus; C, veinal necrosis; D, leaf lamina necrosis.

Discussion

Host-range studies by mechanical inoculations showed that plants of varieties and species representing four genera of the family Convolvulaceae were susceptible to infection by the Feathery Mottle virus. The six varieties of sweetpotato tested were readily infected. Plants of one additional species of the genus Ipomoea, and one species each of the genera Calonyction, Merremia, and Quamoclit proved to be susceptible to the virus.

Many varieties of these species are cultivated and growing wild throughout the South and in the Tropics. The varieties and species tested are a very small part of the total number represented by the family Convolvulaceae. Many of these are perennials and occur as weeds in cultivated fields and adjacent lands. Since some of the members of the wild and cultivated species have been shown to be susceptible to infection by the Feathery Mottle virus, it may be assumed that others also are susceptible if the vector or vectors are present. These plants, particularly the perennials, could prove to be important sources of the Feathery Mottle virus in sweetpotato-growing areas. Once a perennial host plant became infected, it could act as a reservoir of the virus until the area was cropped to sweetpotatoes again under the normal rotation system. Should the Feathery Mottle virus become distributed in infected plants over the sweetpotato-growing areas, these weed hosts could become an important factor in the dissemination and control of the virus in commercial fields as well as in certified- and foundation-seed plots.

Young plants used to grow the sweetpotato crop are ordinarily removed from the plant bed and transplanted to the field at a stage too early for virus infection to be detected (8). Also, under hot, bright conditions during the summer, symptoms of infection are not easily recognized. Plants of Ipomoea purpurea variety Crimson Rambler have given a distinctive reaction when infected by the Feathery Mottle virus. This plant has proved to be a good indicator plant for the virus in sweetpotato plants suspected of containing it (9).

Literature Cited

1. Borders, H. I., and T. J. Ratcliff. 1954. A mosaic of sweetpotato in plant beds and fields in Georgia. Plant Dis. Reptr. 38: 6-9.
2. Doolittle, S. P., and L. L. Harter. 1945. A graft-transmissible virus of sweetpotato. Phytopath. 35: 695-704.
3. Nusbaum, C. J. 1946. Internal cork, a new disease of sweet potato of unidentified cause. Phytopath. 36: 18-23.
4. _____. 1950. Internal cork of sweetpotatoes. South Carolina Agr. Expt. Sta. Bul. 381.
5. Rosen, H. R. 1920. The mosaic disease of sweet potatoes. Arkansas Agr. Expt. Sta. Bul. 167.
6. _____. 1926. The mosaic disease of sweet potatoes with special reference to its transmissibility. Arkansas Agr. Expt. Sta. Bul. 213.
7. Sheffield, F. M. L. 1953. Virus disease of sweet potato in parts of Africa. Empire Jour. Expt. Agr. 21: 184-190.
8. Webb, R. E., and R. H. Larson. 1954. Mechanical and aphid transmission of the feathery mottle virus of sweetpotato. Phytopath. 44: (in press).
9. Webb, R. E. 1954. Morning glory, an indicator plant for the feathery mottle virus. (Abs.) Phytopath. 44: (in press).
10. Weber, G. F. 1923. Mosaic disease of sweet potatoes. Florida Agr. Expt. Sta. Rept. for year ending June 30, 1923. (pp. 59-60).
11. Weimer, James L. 1926. Further evidence of the non-transmissibility of the so-called sweet potato mosaic. (Abs.) Phytopath. 16: 74.

LOUISIANA AGRICULTURAL EXPERIMENT STATION

THE VIRUS DISEASE COMPLEX IN CANNING PEAS[1]

Frank P. McWhorter

Review of Recent Investigations

General surveys of peas in canning areas and specific surveys of fields where field men of the canneries have reported virus diseases have shown, in both 1952 and 1953, that all fields having noticeable amounts of virus diseased plants had more than one virus in most plants. The presence of virus correlated directly with the presence of pea aphids, especially early season pea aphids. Actual trials with the pea aphids (various tests mostly by Clark Amen) showed they were able to transmit all the viruses we have proved to be present in the peas. The complex of viruses in the peas is aphid borne.

It was apparent in 1952 and in 1953 that the pea growers are no longer concerned with a dominant pea virus -- Bean Yellow Mosaic in 1948-49, Enation Mosaic in 1949-50 -- but with a complex of viruses composed of individual viruses having very different infectivity to various varieties of peas. This change, from one virus per pea field to mixtures, has been observed by the field men as various wilts, yellow tops, and especially as tip blights. Thus, former mottles and mosaic symptoms have been succeeded by conspicuous injury to the tops of the plants that prevents late pods from forming.

The virus complex poses new problems with respect to control. We are concerned with the living together of viruses and their combined effects on the peas, and with discovering the hosts that the viruses lived in before the aphids brought them to the peas. First, it was necessary to determine what viruses were present in the peas. That has been done. Also, strains of the viruses concerned have been tested under field and greenhouse conditions to determine the susceptibility of some of the current commercial pea varieties to the viruses.[2] Finally, mixtures of viruses have been tested to determine how living together affects their respective host ranges. This last, as mentioned in this report, may have given us a key to the over-wintering and source of Enation Mosaic Virus, a principal objective of this project.

Current Virus Symptoms

Occasional plants in all pea varieties studied have Enation Mosaic symptoms only: conspicuous white windows or cleared areas in the leaves, rough veins on the leaves, deformed pods. Enation Mosaic is still the commonest virus in the area. Most affected plants, however, show only traces of enation symptoms, the later foliage being wilted, yellowed, spotted as by a fungus, or shrivelled and dried up. Literally, hundreds of tests here have shown that Enation Mosaic alone is not a killer virus in peas, it does not wilt them. The death and yellowing of the tops of the plants is due to the viruses associated with the Enation Mosaic.

Viruses Present in 1953

1) Enation Mosaic Virus combined with Western Ring Spot Virus.
2) Enation Mosaic Virus combined with Alfalfa Mosaic Virus.
3) Enation Mosaic and SG, an undetermined virus.
4) Enation Mosaic-alone (Rare).
5) Alfalfa Mosaic-alone (Rare).
6) Bean Yellow Mosaic -- usually with Ring Spot. (Very rare).
7) Enation Mosaic, Ring Spot, and Alfalfa Mosaic are frequently all present in the same plant.

---

[1]A report to the Canners Operating in Eastern Oregon and Washington. Frank P. McWhorter in cooperation with W. C. Cook. With acknowledgements to H. H. Millsap, W. A. Frazier, John A. Milbrath and Clark Amen whose aid made the experimentation possible.

[2]Many of these tests have been carried out on a very large scale with the cooperation of J. A. Milbrath and W. A. Frazier.

Distribution of Viruses in the Area

The following statements are based on surveys of fields and on analysis of diseased plants sent in by cooperators:

a) The large pea fields in Oregon north of Pendleton have been completely free from all viruses for two years.

b) Bean Yellow Mosaic has been found recently only in peas growing near Walla Walla.

c) Certain fields in all the other areas studied have had the first three combinations of viruses listed above. These areas include Toppenish and newly developed areas in Washington State.

d) As shown below, varieties of peas vary greatly with respect to susceptibility to Western Ring Spot when that virus occurs alone. Unfortunately, it is usually present with other viruses, for example with Bean Yellow Mosaic in Blue Lake Beans and when present with other viruses may be able to infect pea varieties that we consider resistant to Western Ring Spot. Western Ring Spot is certainly general in the area and capable of doing a lot of damage.

e) Alfalfa Mosaic is commonly present in all areas. Unfortunately, there are a multitude of strains of that virus and infectivity to peas varies with the strains. It is very common in peas and almost invariably associated with Enation Mosaic. All the investigations with Dr. Cook indicate that the two viruses must have a common source before they enter the peas. The obvious source is alfalfa. We have many times attempted to find the Enation Mosaic Virus in alfalfa and prove its presence by isolation. That has not been done, but from this season's experiments we have data that indicate Enation Mosaic could overwinter in alfalfa without our being able to detect it.

f) Enation Mosaic is not only the common virus in the pea areas discussed in this report, but is also the virus most commonly present in all the pea growing areas of the Pacific Northwest. A partial explanation of this distribution is the fact that no pea variety is known to be resistant to that virus, its distribution being limited by the migrations of the pea aphids and their preferential choice of pea varieties as they move about in an area.

Effects of These Viruses on Different Varieties of Peas

a) Enation Mosaic: All varieties are considered equally susceptible. Various overgrowth effects are noted as deformations including "no shellum pods." When other viruses are present, the combination stops the terminal growth of the plants.

A virus many times isolated from peas is herein designated "SG". This virus may be a strain of Enation Mosaic Virus that has no visible effect on peas. The "SG" virus has some role in the virus complex that we do not understand.

b) Western Ring Spot: This is an unpublished virus that occurs in legume crops and in potatoes in many areas of the Pacific Northwest. It has always been isolated from plants where it was present in combination with other viruses: with Enation Mosaic in peas, with Bean Yellow Mosaic in beans, with Alfalfa Mosaic in potatoes. In legume crops Western Ring Spot virus behaves somewhat like an activator, its presence being evidenced by far greater injury to the plant than the injury usually credited to the other virus that is obviously present.

The severe top wilts now frequently observed in peas are usually directly related to the presence of this Western Ring Spot Virus. For this reason we have conducted several series of tests to determine the susceptibility of certain pea varieties to this virus. These tests have been made in greenhouse bench plantings and also in exceptionally good field plantings placed for us in a randomized block arrangement by Dr. Frazier. These tests have been made with purified Western Ring Spot Virus, but not with the virus combination with Enation Mosaic. However, we think such tests are a fair measure of the susceptibility of the pea varieties tested. The following statements are supported by data from inoculations of small peas 2 to 3 inches high, and peas in the 6 to 10 inch stage growing vigorously when inoculated, and from records of pea varieties from which the Ring Spot Virus has been isolated.

Susceptibility is inversely proportional to the age of the plants. That is to say the younger the plants when aphids introduce the virus the larger the proportion that will develop Western Ring Spot symptoms. Also, the younger the plants the more they are damaged by the virus. Most small plants of all susceptible varieties die with symptoms like fungus wilt or root rot. Older plants develop apical wilts and necroses, but the plants do not die.

Tabulation of Pea Varieties Tested Extensively for Susceptibility to Western Ring Spot Virus

Highly Susceptible	Highly Resistant
Pefected Wales	Perfected Freezer
Thomas Laxton	Delmar (1952-53 strain)
Alaska	Perfection

The susceptibility of wilt resistant Perfection strains of peas to the Ring Spot virus varies somewhat, but as a group they may be considered resistant. No comprehensive tests of the many strains have been made.

This Western Ring Spot virus has many legume hosts in some of which it induces symptoms that could easily be mistaken for those of Bean Yellow Mosaic. Recent tests indicate that sweetclover is highly susceptible and that many of the symptoms commonly seen in roadside plants in the pea areas may be due to this virus rather than to Bean Yellow Mosaic as we had supposed. Sweetclover is certainly a reservoir plant. Case histories indicate that the virus must be present in alfalfa, but the point has not been proved.

c) Strains of Alfalfa Mosaic Virus: Some twenty strains of Alfalfa Mosaic Virus isolated from alfalfa, potatoes, various clovers, lamb's quarter, physalis, and peas have been tested extensively on type varieties of peas and limitedly on many other varieties. Of special interest are the field tests proving that the fungus-like spotting of the upper foliage frequently seen in pea fields is often due to Alfalfa Mosaic Virus.

The symptoms in peas vary more with the strain of the virus than with the strain of the host inoculated.[3] So great are the variations that peas appear to be most useful test plants to determine strains of Alfalfa Mosaic Virus. Also, very frequently strains of Alfalfa Mosaic Virus can be carried in aphids along with Enation Mosaic, enter the pea plants with that virus, and reproduce in the pea plants, yet fail to produce any detectable symptoms. Thus, the symptoms of Alfalfa Mosaic in peas vary from none to various fungus-like effects on the foliage. Initial symptoms following infection are the shrivelling and dropping of lower leaves of the plant. Premature leaf fall is the only symptom observed for some strains of the virus. Other strains produce noticeable, often prominent, yellowing and fungus-like spotting in the upper foliage. When the tops of naturally infected pea plants are killed, a Ring Spot Virus is usually present also.

Alfalfa Mosaic is a virus that has mutated into countless strains, and, unfortunately for the pea growers, the resistance of peas to the virus varies not with the parent virus, but with the strain. The virus seems able to produce a virus strain adaptable to any pea strain the breeders may produce. However, the resistance of different varieties of peas is amazingly different for a given strain of the virus and only one of the twenty strains tested has been able to infect all of the seven varieties of peas (see ring spot tabulation) that we have inoculated extensively. However, this strain, No. 19, did not infect the pea variety Wando.

It is predicted that some strain of alfalfa mosaic can infect any of our commercial peas, but no single strain will be able to infect all of the commercial varieties. For this report an attempt was made to determine the commonly grown variety most likely to escape infection. No choice could be made.

Of special interest are field inoculations to peas with two strains of Alfalfa Mosaic, OSC strains 19 and 41, originally from potatoes. Strain 19, sent to us by Dr. Menzies from wilted dying potato plants at Prosser, Washington, has the largest range in pea varieties of any strain that we have tested. These two strains were inoculated to "Virus resistant" and unusual varieties of peas being grown by Dr. Frazier in field tests primarily planted to record natural infection by aphid-borne viruses. The varieties, tabulated for infection following inoculation of small plants, are in Table 1.

Bean Yellow Mosaic: This virus has practically disappeared from commercial plantings. The strains that do occur do not infect most varieties with Perfection genes in their constitution.

[3]Delmar is an exception, often showing symptoms resembling those in Ladino clover rather than symptoms in other peas.

Table 1. Infection percentages in pea varieties inoculated with two strains of Alfalfa Mosaic Virus.

Variety	Percentage of infection by alfalfa mosaic virus	
	Strain 19	Strain 41
Geneva Virus Resistant G-168-1	40	25
Dwarf Perfection	75	75
Geneva Virus Resistant G-168-6	35	trace
Freezonian	15	10
PL-140-295	10	5
Geneva Virus Resistant G-168-8	50	70
Wando	none	65

These percentages are based on number of plants showing symptoms, not on the absolute infection determined by recovery of the virus. Nevertheless, they do show that it will be difficult to obtain peas resistant to Alfalfa Mosaic Virus. The injury was rated as a trace to 85 percent, the G-168-8 being the most injured of any in the entire series.

Behavior of Viruses Combined into Complexes

Technically, the most important recent finding of this project is the rediscovery of the circumstance that insects can transmit mixtures of certain viruses that they can not transmit separately. Ring Spot viruses have no known vector. Our observations showed that Ring Spot and Enation viruses were being carried together; that this actually is so was experimentally confirmed by Clark Amen. Our latest discovery, 1953, is that mingling of viruses may extend the host range of a virus.

Three different collections of peas from the Walla Walla area, showing unmistakable Enation Mosaic symptoms, and also yellowing and necrosis characteristic of Western Ring Spot and/or Alfalfa Mosaic viruses, when transferred to Bountiful beans produced a disease that was new to us in beans. Analysis by reliable virus techniques proved that the virus responsible was Enation Mosaic virus. All former studies have indicated that Enation Mosaic cannot go to beans. This statement applies to published accounts over a period of 26 years.

For this record the important point is that the symptoms in beans do not remotely resemble those of Enation Mosaic in peas. It follows, therefore, that Enation Mosaic may be present in other legume hosts (not garden peas, Austrian peas, or sweetpeas) where it produces symptoms so unusual or so different from those in peas that its presence in the new host would be unsuspected. All evidence from field observations indicate that the virus of Enation Mosaic must be present in alfalfa, a circumstance that we have not been able to prove. This finding, that the Enation Mosaic Virus can invade new hosts when combined with other viruses, and produce non-enation symptoms in the new host, indicates that we have been looking for the wrong symptoms in unusual or unknown hosts. These observations, while by no means acceptable as scientific proof, do combine to indicate that alfalfa is not only the home of the pea aphid, but of the Enation Mosaic Virus. Also, they suggest an explanation as to why the current distribution of Enation Mosaic is so erratic. We may suppose that when aphids return to alfalfa, only a small proportion would be carrying a virus mixture capable of introducing the Enation Mosaic Virus into alfalfa. Therefore, only a few alfalfa plants would contain that virus and correspondingly few of the new generation of aphids returning to peas would carry it. Thus, occurrence of the disease would be erratic, as has been the case in the pea fields during the past three years.

The above should be considered a scientific fairy tale that will be subjected to accepted principles of virus analysis during the coming season.

The New Potential Sources of Pea Mosaic

Previous investigations at Washington State College and at the University of Idaho have plainly indicated that a "mosaic" other than Bean Yellow Mosaic occasionally infects peas, including Perfection strains. Such a virus has not been observed during the progress of this investigation. At Oregon State College we are now conducting a comprehensive analysis of the viruses in gladiolus, in which Bean Yellow Mosaic was detected several years ago. That gladiolus viruses can literally ruin a planting of Blue Lake Beans has been abundantly proved. Now, to our surprise we have learned that Bean Yellow Mosaic is not the common mottle virus in gladiolus. Instead, it is a Yellow Mosaic that is properly speaking a pea virus. Thus, the suggestion is made that the former pea mosaics may have been due to viruses that escaped from gladiolus and gradually built up in peas when varieties of peas favorable to that virus were grown.

Gladiolus are not compatible with peas.

Conclusions

1) All recent data from field studies and greenhouse analysis, point to an interrelation of alfalfa and aphids as the explanation of our present virus problems in peas.

2) The current presence of Western Ring Spot and Alfalfa Mosaic viruses emphasizes the desirability of controlling the aphids in young peas by applying insecticides early to prevent the building up of aphid populations.

OREGON AGRICULTURAL EXPERIMENT STATION

POTATO FRECKLES

A. A. Cook

During the summer of 1952, several plants in a field of Bliss Triumph potatoes were found with systemic symptoms unfamiliar to the author. Leaflets of affected plants were somewhat flaccid and distinctly chlorotic except for patches of tissue of the usual green color of healthy potato foliage (Fig. 1). The growth of these plants was slightly reduced but in no other particular were there distinguishable differences. Two such plants were transferred to the greenhouse and plants from the resulting tubers used in preliminary experiments to determine the cause of these symptoms.

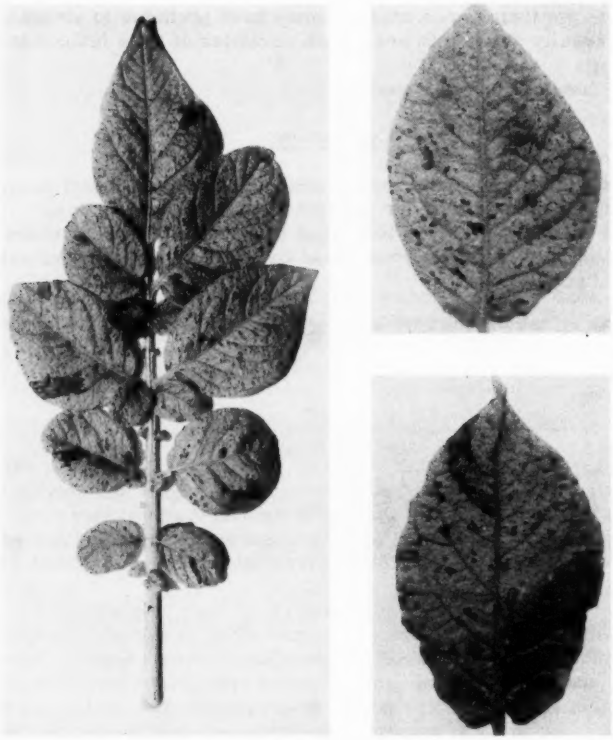


FIGURE 1. "Freckles" on Bliss Triumph potatoes.

Attempts to transmit these symptoms by approach grafting "freckles" plants to plants of the varieties Bliss Triumph, Cherokee, Kennebec, and Seedling 41956 were unsuccessful. Mechanical inoculations to leaves of plants of the above varieties with juice extracted from leaves of affected plants were also unsuccessful. Symptomless plants resulted when the tubers from the above inoculated plants were planted in the field.

Mechanical inoculation to Nicotiana rustica var. brazilia of juice extracted from "freckle" potato leaves produced symptoms indicative of Potato Virus X[1]. Individual lesions of various types were picked out using sharpened veterinary milk tubes and after three to four single lesion transfers onto the above host, and passage through N. glutinosa and Datura stramonium,

[1] Ladeburg, R. C., R. H. Larson and J. C. Walker. 1950. Origin, interrelation and properties of ringspot strains of virus X in American potato varieties. Wis. Agr. Exp. Sta. Res. Bul. 165.

five Virus X strains were isolated. In preliminary cross-protection experiments, each virus strain completely protected against infection by either of the other four.

Inoculation of hybrid seedling potatoes (B76-23 x B61-3) with each of the above virus strains resulted in primary symptoms of necrotic local lesions within five days. Secondary symptoms developed within 14 days and consisted of mosaics and systemic necrotic lesions. The same results were obtained when similar seedlings were inoculated with juice extracted from naturally "freckled" leaves.

The described symptoms on naturally affected potato plants commonly fade and become indistinct as the plants mature. In at least two instances, addition of ammonium nitrate to greenhouse plants induced production of more distinct symptoms.

Although the exact nature of this condition has not been determined, it seems worthy of note that strains of Virus X may be associated with it. Tests for the presence of other viruses, except Potato Viruses Y and A, have not been conducted and cannot, therefore, be eliminated as possible contributing factors. On the basis of this preliminary work the possibility of genetic mutation(s) should also be investigated.

DEPARTMENT OF PLANT PATHOLOGY, SOUTH DAKOTA STATE COLLEGE, BROOKINGS, SOUTH DAKOTA.

THE USE OF VERMICULITE IN PROVIDING SCAB INOCULATION FOR POTATOES

G. V. C. Houghland and L. C. Cash[1]

In conducting studies on the control of scab on potatoes and in investigating factors affecting host susceptibility, it is often necessary to provide suitable inoculation with the scab organism, Streptomyces scabies. In field soils known to be infested with this organism, the infestation is frequently distributed unevenly to the extent that additional inoculation is often considered desirable. In scab studies in the greenhouse, when sand or solution culture methods are used, inoculation must be provided. Moreover, it is sometimes found that field soils known to produce scabby potatoes, when taken into the greenhouse, fail to produce infection unless they are reinoculated.

The method here described is based on the use of vermiculite and Richards' Solution, a medium found satisfactory for growing the scab organism.

Modified Richards' Solution[2]

Potassium nitrate	1.0 gm.
Monopotassium phosphate	.5 gm.
Magnesium sulfate	.2 gm.
Ferric chloride	Trace
Sucrose	5.0 gm.
Distilled water	1,000 ml.

For inoculation purposes in the greenhouse it was found that a sufficient amount of inoculum could be prepared for each plant unit from three tubes of the scab organism growing on potato-dextrose agar slants. To facilitate transferring as much of the growth as possible, about 5 milliliters of water was added to each tube of culture and the agar plugs were removed intact by pulling them out with a wire bent to form a small hook. The growth was cut from the slants and the remainder of the agar discarded. The quantity of this growth to be used was placed in an electric blender, 100 milliliters of water was added for each set of 3 culture tubes transferred, and blending was continued for about 5 minutes.

Estimation of the amount of Richards' Solution required can be made from the amount of inoculum needed. One kilogram of No. 1 vermiculite will absorb approximately 1 liter of solution. To prepare the inoculum, 100 milliliters of the scab suspension was added to 1 liter of Richards' Solution, previously adjusted to pH 7.0 with 0.5 N NaOH, and the 2 solutions were mixed thoroughly. The Richards' Solution scab suspension was then gradually added to 1 kilogram of steam-sterilized vermiculite and mixed thoroughly. This amount of inoculum was sufficient for each plant unit as illustrated in Figure 1. For sand culture use, 1 kilogram of the vermiculite inoculum can be mixed with 5 kilograms of sand to make a mixture that will retain considerable moisture, improve the physical condition of the sand, and at the same time provide inoculation. If the sand culture studies involve nutrient control, water or the nutrient solution to be used can be substituted for Richards' Solution in preparing the inoculum suspension.

The vermiculite-Richards'-Solution inoculum can also be prepared in large quantities for inoculating the soil in a potato field. For this use the inoculum should be held at room temperature for five to six days in order to promote growth of the organism before the inoculum is sown on the land. New growth of the scab organism has been observed on the cut edges of the micaceous plates of the vermiculite six days after the inoculum was prepared. To obtain even distribution in the field, the quantity of inoculum to be applied should be halved and the half portions sown over the entire area in opposite directions. Disking should immediately follow inoculation.

When cultures of Streptomyces scabies are not available, another type of vermiculite inoculum can be prepared from scabby potatoes. Peelings from several hundred pounds of scabby potatoes can be run through an electric meat grinder and the ground pulp mixed with sufficient

---

[1]Biochemist and Assistant Pathologist, respectively, Horticultural Crops Research Branch, Agricultural Research Service, United States Department of Agriculture.

[2]Schaal, L. A. 1944. Variation and physiological specialization in the common scab fungus (Actinomyces scabies). Jour. Agr. Res. 69(5): 169-186.

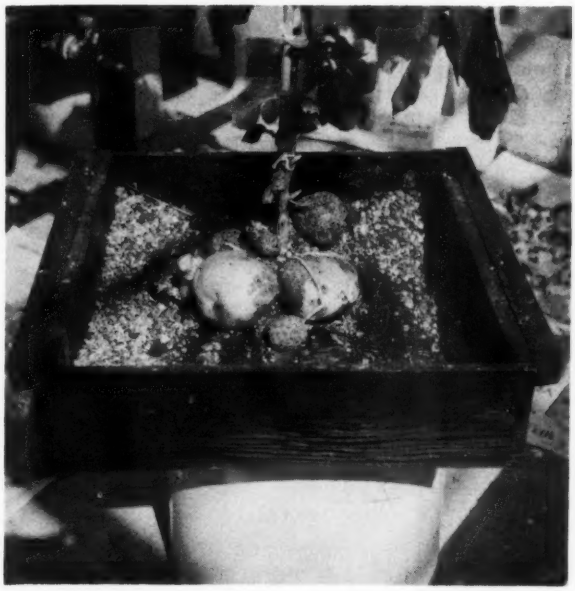


FIGURE 1. Scab infection on potatoes growing in solution culture. Vermiculite inoculum partly removed.

vermiculite to permit easy application. Before either type of inoculum is sown, sufficient lime should be applied to the land, if needed, to bring the soil reaction within the range of pH 5.8 to 7.0.

The procedures described have proved very effective in establishing potato scab in the field and for similar use in the greenhouse. It is probable also that the vermiculite-nutrient-solution method of inoculation may be modified for use with organisms other than Streptomyces scabies.

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE.

VIOLET ROOT ROT OF CELERY[1]

N. J. Whitney[2]

In the fall of 1951, violet root rot of celery, caused by Rhizoctonia crocorum Pers. ex Fr., was discovered on the roots of celery (variety Epicure) growing in the Thedford Marsh in Ontario. In 1952 the disease caused a loss of 7 percent in one field of celery, and again, in 1953, it was responsible for a small amount of damage. Although the disease has been reported on celery grown in England (1) and on the Island of Ely (2), this appears to be the first report of its occurrence in North America (3). Figures 1 to 3 show the effects of violet root rot on some of the plants observed.

FIGURE 1. Left -- a severely diseased plant showing almost complete loss of the root system, and stunting and malformation of the outer leaves. Right -- a healthy plant showing the extent of a normal root system. x approximately 1/4.

R. crocorum attacks the roots of celery in midsummer and by the fall causes almost complete loss of the root system (Fig. 1), which in healthy celery is very extensive. Disease symptoms are not noticeable until early in the fall when the plants begin to wilt and are stunted. The outer leaves turn a yellowish color and the petioles, which are normally crisp and green at this time of the year, become faded and spongy. When the petioles of such leaves are cut they appear dry and stringy. When the diseased plant is lifted, mycelium and infection bodies of R. crocorum can be seen completely covering the roots remaining on badly diseased plants, and in patches on the roots of plants only slightly diseased (Fig. 2). As is typical of this fungus

---

[1]Contribution No. 1392 from the Botany and Plant Pathology Division, Science Service, Department of Agriculture, Ottawa, Canada.

[2]Associate Plant Pathologist.

FIGURE 2. (Two diseased plants). Left -- plant moderately diseased. Much of the fibrous root is still intact. Right -- plant severely diseased. The blackened roots denote complete infection by R. crocorum. Note the faded appearance of the foliage. x approximately 1/4.

FIGURE 3. A celery leaf showing a mass of R. crocorum growing around the base of the petiole. x 1/4.

on the roots of other hosts, a purplish mycelial weft covers the root presenting a characteristic violaceous discoloration. Beneath this mycelial mat there are located in the infected root tissue numerous minute infection bodies which arise from the infected tissue. Late in the fall, the fungus can be found growing a short distance up the leaf petioles (Fig. 3), but it does not appear to be parasitic on the leaf tissues. R. crocorum was easily isolated from diseased roots by plating out on malt agar a thin section of infected tissue containing mature infection bodies.

In the Thedford Marsh both early and late celery are grown. The early celery is planted in May and the late celery in the latter part of June or early July. Usually both crops are harvested during October. So far, although slight infection has been encountered on the late celery, only the early celery has been badly diseased. As is the case with carrots, susceptibility of celery to violet root rot seems to depend upon the age of the plant[3]. In severe cases of violet root rot the leaf petioles become dried out because of the loss of the root system at the time when the plant is normally maturing. The infected plants are therefore unmarketable and represent a complete loss to the grower.

Literature Cited

1. Brit. Min. Agr. & Fish. Bull. 79: 56. 1934.
2. Brit. Min. Agr. & Fish. Bull. 139: 44. 1948.
3. Conners, I. L., and D. B. O. Savile. 1953. Canadian Plant Disease Survey (1952). Ann. Rept. 32: 47.

SCIENCE SERVICE LABORATORY, HARROW, ONTARIO.

[3] Author's unpublished data.

WRINKLED SEED COAT DEFECT IN GREEN LIMA BEANS FOR FREEZING

W. B. Neuburg[2], John H. MacGillivray[3], Mas Yamaguchi[3], and Louis K. Mann[3]

During the last two years there has appeared in lima beans a seed coat or testa deformity that has been responsible for lowering the grade of the frozen product. This deformity causes a small area of the interior layer of the seed coat to turn brown after processing and cooking. The discoloration of the seed coat next to the cotyledon is evident on both raw (Fig. 1) and blanched beans, but only after blanching or cooking does the discoloration show on the outside of the seed coat. None of the green cotyledon is affected. This defect is difficult to detect and consequently hand-sorting of the blanched beans on factory belts is not practical. It has occurred in only a few fields primarily in Santa Clara and Monterey Counties, in California. While other areas of California have not been free of wrinkled beans, they have experienced little economic loss. U. S. Grade A frozen beans can only have 2 percent of wrinkled beans, or about four per package, and still comply with the grade. This condition has received various names, including pebbled, blister, wrinkled, alligator hide, and brown spot:

Wrinkled beans are found most frequently on plants that are somewhat stunted in growth. The Concentrated Fordhook has been the only variety to exhibit any appreciable amount of this defect. Since this wrinkling cannot be detected from the appearance of the pod, it is necessary to open the pod and examine the beans. Consequently, processors' field men find it difficult to identify affected areas within a field. The defective area of the bean is usually wrinkled on the exterior surface. When the seed coat, or testa, is split the inside surface appears cracked and is yellowish-brown in color. Occasionally, pods are opened in which the cotyledons have burst the seed coats. In only a few cases has this defect been found near the hilum. It is generally more common near the outer circumference of the bean. The cracked condition affects the layers of cells near the green cotyledon and does not extend through the seed coat to the outer surface of the unblanched seeds. The affected beans are more likely to be located away from the place of attachment to the plant. Sometimes all the beans in a pod are affected but most frequently only one or two. Half-grown beans show this defect less frequently than beans that are at the mature green stage for freezing. The best way to check this condition in the green beans is to look for the wrinkled condition and then examine the inside of the seed coat. The brown area on the seed coat can best be demonstrated by placing the beans in boiling water for eight to ten minutes.

Thin sections from affected beans were stained and examined microscopically to determine precisely the region of the seed covering that becomes discolored. The dark tissue showing injury, except where the whole seed covering is split, is confined to the inner surface of the covering. The exact nature of the affected layer cannot be determined without study of younger seeds, but it appears to be the remains of a nutritive tissue known as endosperm. It is this layer and not the integuments on the cotyledons that disorganizes, cracks, and discolors.

The disorganized cells form a rather homogeneous dark-staining material that sticks to the surface of the cotyledons and may penetrate outward into the intercellular spaces of the seed coat cells. This material forms minute knobs and protuberances on the cell walls. The somewhat disorganized regions in the integuments or seed-coat proper are probably areas where material from the disorganized endosperm has penetrated. It should be emphasized that a study of all sizes of seeds must be made to confirm the above interpretation of seed coat layers.

Wrinkled seed coat was observed during August, 1952 in a field that was undoubtedly suffering from a water shortage. This was indicated by the appearance of the plants and soil as well as by the remarks of the grower. It seems likely that the defect was caused by poor growing conditions during the last third of the growing season. Since soil moisture is such an important factor in plant growth, it may well be the major causal agent. High temperatures have also been suggested as a cause. During such periods soil moisture is more likely to be a limiting factor. Early fields are more apt to have this defect, and usually wrinkling is more severe on the plants from the poorer areas of the field. Cases have been known where 15 to 30 percent of the beans in one part of a field may show this defect. Other parts of the same field may be only slightly affected. Analyses of normal and affected seed coats for major or minor elements have not re-

[1]The authors wish to express their appreciation to L. J. Clemente, Robert Enparan, and Arthur Greathead as well as Ed McCandless and William Schlapia of Spiegl Foods for assistance and interest in this problem.

[2]Horticulturist, Birds Eye Division, General Foods Corporation, Hillsboro, Oregon.
[3]Department of Vegetable Crops, University of California, Davis, California.

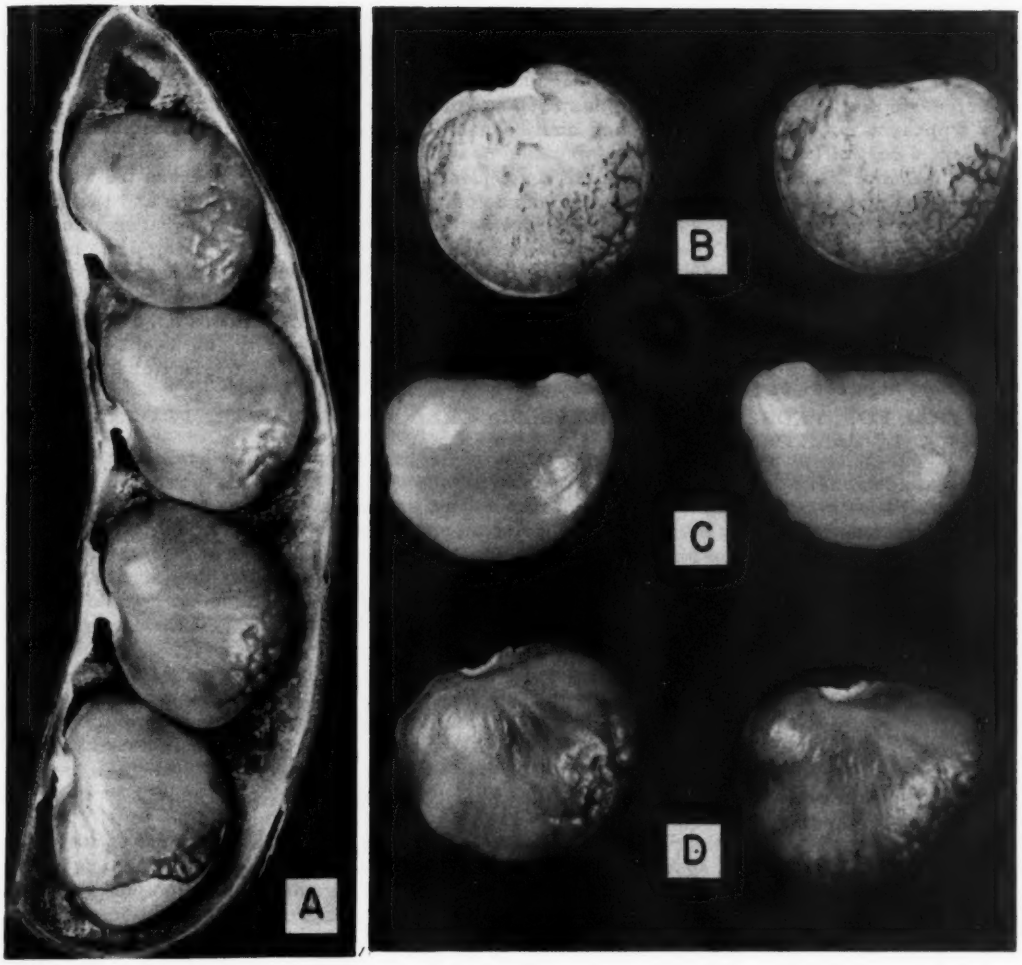


FIGURE 1, Wrinkled defect in green lima beans. A -- Pod with beans showing wrinkled defect and a cotyledon which has burst the seed coat. B -- The inside of the removed seed coat showing cracked condition caused by defect. C -- Cotyledon removed from seed coat. D -- Beans before removal of seed coat showing wrinkled defect on lower margin.

vealed very striking differences. Likewise, fertilizer experiments in Santa Clara County did not indicate any important relationships to soil nutrients. These same experiments included three irrigation treatments -- normal and with one and two irrigations omitted late in the season. In all cases the omission of two irrigations increased the percentage of wrinkled beans. However, on the lightest soil even the normal irrigation gave 3.4 percent wrinkled beans, whereas when two irrigations were omitted 8.8 percent of the beans were affected. These data indicate a relationship between the relative amount of this defect and irrigation treatment.

Wrinkled beans are not commonly found throughout California green lima bean areas. In some parts of a few fields this defect has been so severe that it was impossible to comply with the standards for U. S. Grade A frozen lima beans. There are two sources of economic loss -- the added cost of trying to sort out the wrinkled beans at the processing plant and the abandoning of fields or sections of a field with the consequent loss to the grower. Where the wrinkled beans reach the freezing plant there is the extra cost of slowing plant operation and putting on extra help to try to pick out defective beans, as well as the loss due to lower grades with resulting lower sale value. Cases are known where the economic loss to the factory has been $30,000 to $45,000 per season. Since most of the difficulty has been with Concentrated Fordhook, it would seem desirable to use the U. S. 242 variety in less favorable climates and on marginal soils low in water-holding capacity. Unfortunately, U. S. 242 does not always yield so well and the plant is larger. Since the defect increases with maturity, the beans should be harvested just before or at their peak quality and not allowed to become overmature.

GENERAL FOODS CORPORATION, HILLSBORO, OREGON, AND UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA.

FIELD TESTS WITH CHEMICALS FOR THE CONTROL OF RHIZOCTONIA AND OTHER PATHOGENS OF COTTON SEEDLINGS

L. A. Brinkerhoff, E. S. Oswalt, and J. F. Tomlinson[1, 2]

SUMMARY

The results from tests that were artificially infested with Rhizoctonia solani indicated that this fungus may cause severe seed decay and pre-emergence injury as well as damping off and sore shin.

Pentachloronitrobenzene applied to the soil in the seed row was effective against R. solani in artificially infested tests, but failed to control seed decay and pre-emergence injury during cold, wet weather in naturally infested tests.

Thiram, on the other hand, was effective when PCNB failed. It would appear that perhaps a pathogen other than R. solani was responsible for these differences. Pythium sp. has recently been isolated from cotton seed in cold soil, but pathogenicity tests have not yet been completed.

Seed treatment, especially with slurry preparations of thiram (Arasan SF-X and Thiram Naugets), appeared to give reasonably good protection against the seed decay and pre-emergence injury that occurred during cool, wet weather. This was indicated in the seed treatment tests at Chickasha in 1952, the May 11 planting at Stillwater in 1953, and the test on the Robert Lowe Farm at Chickasha in 1953.

Fuzzy seed produced significantly better emergence during cool, wet weather, but acid-delinted seed were superior when temperatures favored fairly rapid germination.

The data from tests artificially infested with R. solani, some of which are not reported here, indicate that seed treatment has considerable value in protecting cotton seed from injury caused by R. solani. However, when the inoculum level was high or the environment favorable for post-emergence injury, seed treatment did not give adequate protection to insure satisfactory stands. Results similar to these also were obtained in the greenhouse.

The same isolate of R. solani was pathogenic over a wide temperature range, but appeared to be more destructive at relatively cool temperatures (average daily air temperatures of 65° to 75° F), especially when accompanied by high soil moisture and high relative humidity. Epiphytotics caused by R. solani and possibly other pathogens occurred in cotton following green manure crops of winter rye and vetch in Central and Eastern Oklahoma in 1950 and 1951 during relatively cool, wet weather, but not in 1952 and 1953 when rainfall was sparse and temperatures high.

During the past two seasons an attempt has been made to obtain more effective control of seedling diseases of cotton in Oklahoma. Several chemicals have been used as seed protectants and soil fungicides in both hand- and machine-planted tests. Plots were artificially infested with Rhizoctonia solani for a number of the tests.

The results are considered tentative, but are presented here for the consideration of other investigators who are working on related problems.

1952 TESTS

HAND PLANTED TESTS AT CHICKASHA, OKLAHOMA: Rows for the hand planted tests were laid out with a 4-row lister planter which was set to make a shallow furrow 3 to 4 inches deep. Each plot consisted of a single row 33 feet long planted with 100 seed hill dropped.

---

[1]Respectively, Plant Pathologist, Field Crops Research Branch, Agricultural Research Service, USDA, and Botany and Plant Pathology Department, Oklahoma A. & M. College; Superintendent, Oklahoma Cotton Research Station, Chickasha, Oklahoma; and Farm Co-ordinator, Oklahoma Agricultural Experiment Station.

[2]Acknowledgement is made to Mr. A. M. Hillis of the Mathieson Chemical Corp. and to Dr. M. J. Sloan of Shell Chemical Corp. for assistance in planting tests at Tipton and Chickasha in 1953. Acknowledgement is also made to Mr. Robert Lowe and to Mr. Leo Smith, who served as cooperators.

The furrows for the seed were made in the bottom of the lister rows and were covered with a Planet Jr. seeder with openers removed. Hills were marked by lugs on the wheel of a small garden plow, and the seed were dropped by hand.

Plots were artificially infested with an isolate of R. solani obtained from a diseased cotton seedling. The same isolate was used for all tests. Inoculum was grown on steamed grain sorghum and chopped in a food blender, after which it was strained through cheese cloth so that the resulting hyphal suspension was fine enough to pass through an ordinary sprinkling can. Stock suspensions were prepared just prior to use by diluting the contents of each 250 ml. flask of inoculum with 2 liters of water. Each plot received 200 mls. of the stock suspension that was further diluted with 3 pints of water and applied to the open row just before the seed were planted.

Seed for the tests were from a lot of Stoneville 62-1 which germinated 77 percent in sterilized soil for the fuzzy seed and 89 percent for the heavy fraction of gravity graded acid delinted seed.

Test 1. -- Seven chemicals were compared as drill-row fungicides at 1 and 2 pounds per acre (calculated on the basis of 40-inch rows) in a randomized block design with 9 replicates. Dow 9B, Spergon, Arasan, Ceresan M, and Systox were mixed with dry sand and applied at the rate of 1 pound of sand per plot in a 4 to 5 inch band in the open row before the seed was covered. The two liquid materials, Setrete and Vancide 51, were diluted with 1 quart of water and applied with a sprinkling can. Acid-delinted (heavy) seed were used throughout and treated with Spergon Sl except for a nontreated check. All plots received inoculum.

Test 2. -- This was a seed treatment test in which non-treated seed and four chemicals were compared on both fuzzy and acid-delinted (heavy) seed. Spergon Sl, Ceresan M, and Arasan SF-X were used as slurry preparations and Setrete as a liquid. All plots received inoculum. The planting design was a 10 x 10 latin square.

Test 3. -- Artificially infested plots and non-infested plots were compared in a randomized block design with 9 replicates. Agrox and 5-10-10 fertilizer, each as drill-row treatments, were included as additional variables. Treated seed were used only for the Agrox drill-row treatment. The seed treatment chemical was Spergon.

Test 4. -- This test occupied the same area as Test 1, which had been disked under because of a poor stand. Arasan, Dow 9B, and Spergon were compared as seed and drill-row treatments in a randomized block design with 4 replicates. In each instance the seed were treated with the same chemical as used in the drill row. Other variables included kinds of seed and use of inoculum. Instead of applying all of the chemicals to the open row as previously, one-half was applied after the seed had been partially covered.

Tests 1 and 2 were planted on May 10, Test 3 May 14, and Test 4 June 6. Heavy rains accompanied by relatively cool temperatures occurred between May 15 and May 24. Air temperatures (average of daily maximum and minimum readings) and rainfall are shown in Figure 1. Temperatures favored rapid germination for Test 4, and there was no rain while the seedlings were emerging.

Results. -- During the first three tests the weather was especially favorable for seed rot and pre-emergence injury. Test 3 was the most severely affected; even the plots which did not receive inoculum were as severely affected as the ones where inoculum was applied. These plots averaged only 4 seedlings per 100 seeds.

In Test 1 final stands in plots treated with chemicals in the drill row were no better than those in which only the seed were treated. Seed treated with Spergon averaged only 19.3 seedlings per 100 seeds, whereas non-treated seed averaged 14.3 seedlings. This difference was statistically significant.

In the seed treatment test, Arasan SF-X produced substantial increases in stand with both acid-delinted and fuzzy seed. Fuzzy seed averaged more seedlings and somewhat higher yields, notwithstanding a 12 percent advantage in initial germination for the acid-delinted seed. Comparisons of stand and yields are in Table 1.

Emergence, with seed of the same lot as used for the previous tests, was much higher in Test 4. Inoculation reduced both stands and yields, but the differences were relatively small. Again, seed treatments were significantly better than no treatment, but drill-row applications were not. There were no appreciable differences among chemicals, although Arasan SF-X was somewhat better where inoculum was not applied to the soil. Under the conditions of this test

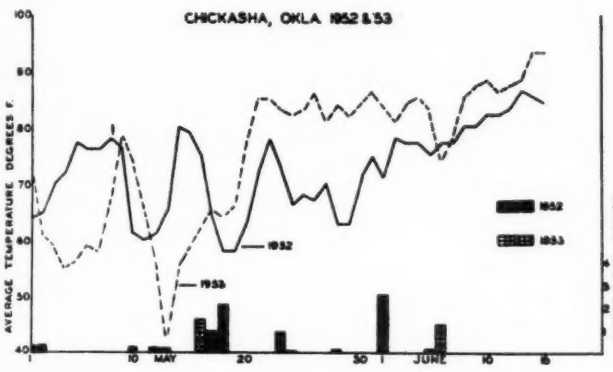


FIGURE 1. Average daily air temperatures (average of maximum and minimum) and daily rainfall at Chickasha, Oklahoma.

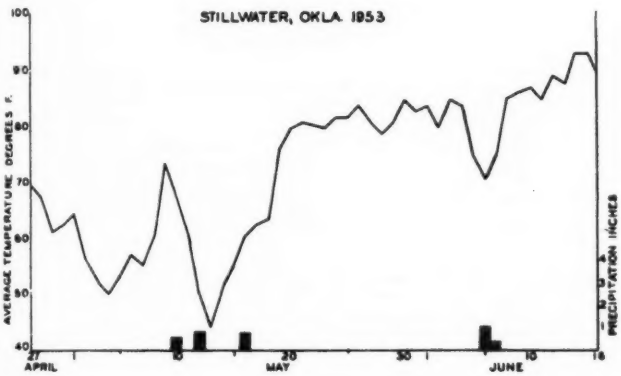


FIGURE 2. Average daily air temperatures (average of maximum and minimum) and daily rainfall at Stillwater, Oklahoma.

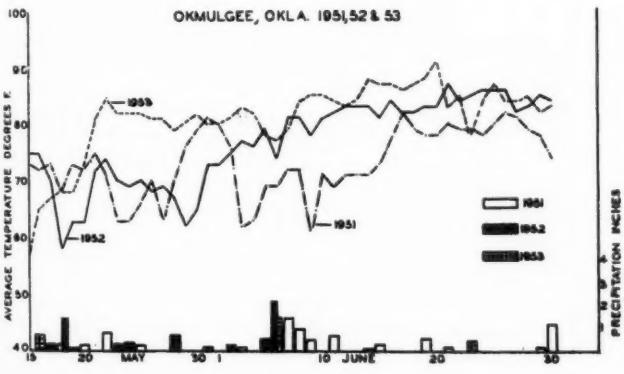


FIGURE 3. Average daily air temperatures (average of maximum and minimum) and daily rainfall at Okmulgee, Oklahoma.

Table 1. A comparison of four seed protectants on fuzzy and an acid-delinted cotton seed in a field test in which inoculum of Rhizoctonia solani was applied to the drill row. Planted May 10, 1952, Cotton Research Station, Chickasha, Oklahoma.

Kind of seed and chemical treatment	Surviving seedlings after 26 days (percent)	Yield of lint per acre (pounds)
Fuzzy		
Spergon Sl.	28.1	295
Ceresan M Sl.	26.6	281
Setrete	26.7	249
Arasan SF-X	41.8	294
Check	22.3	232
Acid-delinted		
Spergon Sl.	20.5	249
Ceresan M Sl.	20.2	232
Setrete	20.9	242
Arasan SF-X	33.3	334
Check	10.3	167
L.S.D. at the 5% level	7.3	75
Average for fuzzy	29.1	270
Average for acid-delinted	21.0	244
L.S.D. at the 5% level	3.3	33

acid-delinted seed was superior to fuzzy seed in both stands and yields. Data for Test 4 are in Table 2.

MACHINE PLANTED TESTS: Two tests were made near Okmulgee, Oklahoma, following a winter crop of rye and vetch which had been plowed under about three weeks at the time the cotton was planted. During the two previous seasons, when considerable rain occurred after planting, post-emergence damage from R. solani had been very severe in this area. Seasonal differences in temperature and rainfall for the three years are shown in Figure 3.

Arasan as a 50% dust was mixed with 5-10-10 powdered fertilizer and applied at the rate of 1, 2, and 4 lbs. of Arasan and 150 lbs. of fertilizer per acre. The fertilizer-fungicide mixture was applied to the drill row with the applicator spout set so that the chemicals were mixed with the upper two-thirds of the soil that covered the seed.

Results. -- The weather did not favor disease development (see Fig. 3). No evidence of phytotoxicity was observed.

1953 TESTS

HAND PLANTED DRILL-ROW TESTS: A test at Stillwater compared Arasan SF-X and Ceresan M as slurry seed treatments and Thiram 50 and Mathieson 275 (pentachloronitrobenzene) as drill-row fungicides. Seed for the drill-row comparisons were treated with Arasan SF-X (slurry). Other variables included: date of planting (April 27, May 11, and June 9), kinds of seed (fuzzy and acid-delinted), and inoculation. The method of planting and infesting the soil was similar to that followed in 1952, except that the drill-row chemicals were applied by means of a funnel and spout attached to the Planet Jr. seeder used for covering. The chemical-sand mixture was reasonably well mixed with soil that covered the seed.

In a second test, planted at Chickasha on May 21, 6 chemicals were compared at 3 rates in the drill row in soil artificially infested with R. solani. Seed were treated with Arasan SF-X applied as a slurry. Checks were included to determine the effect of seed treatment, inoculation,

Table 2. Surviving seedlings and yields of a hand planted field test comparing chemicals, method of treating, kind of seed, and artificial inoculation. Planted June 6, 1952. Cotton Research Station, Chickasha, Oklahoma.

Variables[a]	Percent of seedlings Emerged (1 week)	Percent of seedlings Surviving (3 weeks)	Yield of lint lbs./acre
Inoculation with R. solani			
Inoculated (all treatments)	64.0	60.3	116
Not inoculated (all treatments)	68.0	66.5	121
Kind of seed			
Acid-delinted (all treatments)	69.6	67.2	125
Fuzzy (all treatments)	62.4	59.6	113
Methods of treating			
Seed treatment	71.7	68.5	124
Seed treatment and row treatment	65.8	64.7	122
Chemicals			
Arasan	70.1	68.3	130
Dow 9B	67.5	65.9	116
Spergon	67.8	65.6	122
None-check	57.8	53.8	108
Checks -- No chemicals			
Inoculated, acid delinted	58.4	52.1	109
Inoculated, fuzzy	48.9	43.3	87
Not inoculated, acid delinted	68.9	67.4	121
Not inoculated, fuzzy	54.9	52.2	111

[a]The analysis of variance for surviving seedlings showed significance at the 1 percent level for kinds of seed, inoculation, and chemicals but not for methods of treating.

and use of vermiculite as a soil conditioner. The inoculum was not macerated as previously, but applied as whole kernels of a grain sorghum culture. Two kernels of inoculum were planted with each hill of cotton seed. The vermiculite was used as a carrier for the chemicals at the rate of 1 1/2 pints for a 33-foot plot, and was applied in a 4 to 5 inch band to the open row just prior to covering.

The variety Deltapine 15 was used for the former test and Stoneville 62 for the latter.

Results. -- Data for the April 27 planting of the Stillwater test are in Table 3. Seedlings emerged after 6 to 10 days. Germination was adversely affected by winds which dried the soil to the seed level in some instances. Under these conditions acid-delinted (heavy) seed gave better emergence except for non-treated seed in plots infested with R. solani. Severe post-emergence injury occurred in both inoculated and non-inoculated blocks and was associated with a period of cold, rainy weather (see Fig. 2).

Rainy weather permitted only one replicate of the May 11 Stillwater planting to be made. This block was not artificially infested with R. solani. The results (see Table 4) are similar to those obtained at Chickasha in both 1952 and 1953 during periods of similar weather (see Tables 1 and 7). The injury occurred primarily as seed decay and pre-emergence damage. The third planting at Stillwater was adversely affected by extremely hot weather.

Unseasonably warm weather favored rapid germination in the May 21 planting at Chickasha. Seedlings emerged within 4 to 6 days. R. solani was extremely pathogenic in plots infested with whole grain inoculum, the injury occurring primarily as pre-emergence damage. Seed treatment with Arasan SF-X was not effective with this type of inoculum.

Drill-row applications of PCNB (Mathieson 275 and Chipman 3143) at 4 and 8 lbs. per acre gave reasonably good stands with whole grain inoculum (see Table 5). The poor results obtained with the remaining chemicals may have resulted from phytotoxicity rather than failure to control

Table 3. Data obtained for chemicals applied as seed and drill-row fungicides for the control of cotton seedling diseases.[a]

Treatment	Rate		R. solani inoculum added: Cotton seedlings			No inoculum added: Cotton seedlings		
	gms./kg.	lbs./A.	Emerged %	Surviving %	Killed %	Emerged %	Surviving %	Killed %
Fuzzy Seed								
Seed treatment								
Check (None)	-	-	21.3	7.3	65.7	40.0	25.6	36.0
Arasan SF-X	3	-	52.3	34.3	34.4	54.6	46.3	14.8
Ceresan M	3	-	51.9	27.0	48.0	55.9	34.6	38.1
Seed treatment + drill row								
Arasan SF-X + Thiram (50%)	3	4	50.3	39.0	22.5	50.9	37.6	26.1
Arasan SF-X + Math. 275 (50%)	3	8	54.9	32.3	41.2	56.6	43.6	23.0
Mean for fuzzy seed			46.2	28.0	39.4	51.7	37.6	27.3
Acid-Delinted Seed								
Seed treatment								
Check (None)	-	-	24.6	12.0	51.2	41.6	15.3	63.2
Arasan SF-X	3	-	66.9	33.3	50.2	73.9	45.9	37.9
Ceresan M	3	-	74.6	43.3	42.0	72.2	51.6	28.5
Seed treatment + drill row								
Arasan SF-X + Thiram (50%)	3	4	73.6	47.3	35.7	76.3	56.6	25.8
Arasan SF-X + Math. 275 (50%)	3	8	68.9	44.0	36.1	62.9	40.6	35.5
Mean for acid-delinted seed			61.8	37.0	40.1	65.5	42.1	35.7

[a]Hand planted field test, West Agronomy Farm, Stillwater, Oklahoma, planted April 27, 1953.

R. solani. Unfortunately non-inoculated plots were not treated with chemicals for comparison.

MACHINE PLANTED TESTS: Two plantings were made in which whole grain inoculum was applied with the seed at the rate of 1 part of inoculum to 10 parts of seeds. A test at Chickasha was planted on May 6 and another at Tipton on May 7. Shell CBP (chlorobromopropene) was applied as a liquid spray directed into the drill row after the seed were partially covered, and Mathieson 275 was blown into the drill row as a dust from two flexible tubes, one of which was directed into the open row and the other into the partially covered row.

An additional test was planted in the vicinity of Chickasha on Mr. Robert Lowe's farm in which chemicals were applied only as dusts. The field had been cropped with a winter cover crop of wheat and Austrian peas. Artificial inoculum was not used nor were the treatments randomized.

The three tests were made just prior to a period of cold, rainy weather (see Fig. 1). The weather data for Tipton were very similar to those for Chickasha, and are not shown.

Non-treated acid-delinted seed were used for the Chickasha test, non-treated fuzzy seed at Tipton, while acid-delinted Ceresan M treated seed were used in the third test.

Results. -- Pre-emergence injury was extremely severe in the artificially infested checks at Tipton and Chickasha. Non-inoculated checks produced only about 1 seedling for each 4 seeds planted. Mathieson 275 dust (20% active) at 20 and 64 lbs. per acre (on a 40-inch row basis) controlled both pre- and post-emergence injury at Tipton, but was not effective at the 14 lbs. per acre rate in the test at Chickasha. CBP appeared to be phytotoxic in both tests and may

Table 4. Surviving seedlings for three dates of planting in a hand planted field test comparing chemicals, methods of treating, kinds of seeds, and artificial inoculation with R. solani.

Treatment	Rate: Seed (gms./kg.)	Rate: Soil (lbs./acre)	Surviving seedlings (percent): 1st planting[a] Inoc.	1st planting[a] Check	2nd planting[b]	3rd planting[c] Inoc.	3rd planting[c] Check
Fuzzy Seed							
Seed treatment							
Check (None)	-	-	7.3	25.6	31	13.3	20.3
Arasan SF-X	3	-	34.3	46.3	61	10.7	14.7
Ceresan M	3	-	37.0	34.6	53	17.7	27.6
Seed treatment + drill row							
Arasan SF-X + Thiram 50 (50%)	3	4	39.0	37.6	74	14.3	24.3
Arasan SF-X + Math. 275 (50%)	3	8	32.3	43.6	41	11.3	13.3
Mean for fuzzy seed			28.0	37.6	52.2	13.5	20.1
Acid-Delinted							
Seed treatment							
Check (None)	-	-	12.0	15.3	3	15.3	22.6
Arasan SF-X	3	-	33.3	45.9	33	16.7	30.0
Ceresan M	3	-	43.3	51.6	48	25.6	31.0
Seed treatment + drill row							
Arasan SF-X + Thiram 50	3	4	47.3	56.6	54	28.0	29.3
Arasan SF-X + Math. 275 (50%)	3	8	44.0	40.6	33	21.3	23.0
Mean for acid delinted seed			37.0	42.1	34.2	21.4	27.2

[a]Planted April 27, 1953.
[b]Planted May 11, 1953.
[c]Planted June 9, 1953.

have been applied too close to the seed. The relative number of surviving seedlings for both tests are listed in Table 6.

Severe seed decay occurred in the test at Chickasha following the green manure crop (see Table 7). Mathieson 275 was not effective although the rates of application were comparable to those used at Tipton. Reasonably good control, however, was obtained with Thiram 50 as a dust in the drill row at 13 and 27 lbs. per acre, and also with Thiram Naugets used only as a slurry preparation on the seed at 6 gms. per kg. of seed. Arasan dust at a comparable dosage was not nearly so effective as the slurry seed treatment with Thiram Naugets. Dithane Z-78 at 14 lbs. per acre likewise was not so effective as seed treatment with Thiram Naugets.

TESTS WITH FUNGICIDES APPLIED WITH FERTILIZER: Mathieson 275 (50% active) at 5 and 15 lbs. per acre, and Dithane Z-78 (20%) at 15 lbs. were each mixed with 10-20-10 fertilizer and applied to the soil covering the seed as in 1952. The planting was made on May 27. Relatively little seed decay or seedling injury occurred although there was an abundance of non-decomposed organic matter present at planting time. The weather favored rapid germination of the cotton seed (see Fig. 3). A light gentle rain after planting insured moisture for germination, but high temperatures, with almost no additional rain, then prevailed for several weeks. There was no evidence of phytotoxicity with either of the chemicals.

Table 5. Hand planted test comparing chemicals for the control of injury to cotton seed and seedlings caused by R. solani[a].

Treatment[b]	Rate (Chemical treatments)	Seedlings Emerged (1 week) %	Seedlings Surviving (7 weeks) %
Drill row + seed treatment			
Chipman 3142 (50%) + inoc.	4 lbs/acre	41.5	27.8
Chipman 3142 (50%) + inoc.	8 lbs/acre	49.3	38.8
Chipman 3143 (50%) + inoc.	4 lbs/acre	47.3	44.8
Chipman 3143 (50%) + inoc.	8 lbs/acre	57.1	58.3
Mathieson 275 (50%) + inoc.	4 lbs/acre	62.5	61.0
Mathieson 275 (50%) + Inoc.	8 lbs/acre	61.5	64.3
Thiram 50 (50%) + inoc.	3 lbs/acre	20.7	9.0
Thiram 50 (50%) + inoc.	6 lbs/acre	19.8	12.0
Penn Salt NP1083 (25%) + inoc.	4 lbs/acre	6.8	10.0
Penn Salt NP1083 (25%) + inoc.	8 lbs/acre	8.7	9.5
Dithane Z-78 (20%) + inoc.	8 lbs/acre	13.2	6.7
Dithane Z-78 (20%) + inoc.	16 lbs/acre	18.2	8.8
Seed treatment + Carrier			
Arasan SF-X, + Vermiculite, inoc.	3 gms/Kg	8.2	4.8
Arasan SF-X, + No vermic., inoc.	3 gms/Kg	8.0	3.8
Arasan SF-X, + Vermic., No inoc.	3 gms/Kg	81.6	81.7
Arasan SF-X, + No vermic., No inoc.	3 gms/Kg	78.8	86.5
No seed treat., + Vermic., No inoc.		78.8	83.2

[a]Cotton Research Station, Chickasha, Okla., May 21, 1953.
[b]The active fungicides are: Chipman 3142, tetrachloro-nitrobenzene; Chipman 3143 and Mathieson 275, pentachloronitrobenzene; Thiram 50, tetramethyl-thiruramdisulfide; Penn Salt NP-1083, 2, 4-dinitro-fluorobenzene; Dithane Z-78, zinc ethylene-bisdithiocarbamate.

Table 6. Machine planted field tests comparing drill row applications of Mathieson 275 and Shell CBP for the control of injury caused by R. solani.

Treatment	Rate	Surviving seedlings per 100 ft. of row
	Test 1 (Chickasha, Okla., May 6 to May 26, 1953)	
Drill row only		
Mathieson 275 (20% dust)	14 lbs./acre	45.8
Shell CBP (38%) (liquid spray)	1.1 qts./acre[a]	22.1
Check (inoculated only)	--	44.0
Check (no inoculum)	--	104.6
	Test 2 (Tipton, Okla., May 7 to June 3, 1953)	
Drill row only		
Mathieson 275 (20% dust)	20 lbs./acre	103.6
Check (inoculated)	--	17.0
Mathieson 275 (20% dust)	64 lbs./acre	105.2
Check (inoculated only)	--	20.3
Shell CBP (38%) liquid spray	0.8 qts./acre[a]	5.7
Check (inoculated)	--	5.8
Shell CBP (38%) liquid spray	2.1 qts./acre[a]	2.0
Check (inoculated)	--	11.0
Check (not inoculated)	--	70.5

[a]Applied in 4 gal. of water.

Table 7. Comparison of chemicals as seed treatments and dusts applied to the drill row for the control of cotton seedling diseases. Robert Lowe Farm, Chickasha, Okla., Planted May 11, 1953.

Treatment	Rate		Surviving Seedlings per 100 ft. of row
	gms./kg.	lbs./acre	
Seed treated with Arasan dust	6	--	3.4
Drill row-Math. 275 (20%) dust	-	19	14.7
Seed treat. -- original Ceresan M only (check)	6	--	35.7
Drill row -- Math. (20%) + Arasan dust seed treatment	6	20	39.7
Seed treated with Thiram Naugets slurry	6	--	112.2
Drill row -- Math. 275 (50%)	6	26	19.7
Seed treated with Thiram Naugets slurry	6	--	91.7
Drill row -- Math. 275 (50%) + Thiram N. Seed treat.	-	12	53.8
Seed treated with additional Ceresan M.	2	--	63.8
Drill row -- Dithane Z-78 (20%) + Thiram N. sl. seed treat.	6	27	105.4
Seed treated with Thiram Naugets slurry	6	--	90.5
Drill row -- Dithane Z-78 (20%)	6	14	49.3
Seed treated with Thiram Naugets slurry	6	--	53.8
Drill row -- Mathieson (50%)	6	27	23.4
Seed treated with Thiram Naugets slurry	6	--	103.7
Drill row -- Mathieson (50%)	-	8	19.8
Seed treated with Thiram Naugets slurry	6	--	110.4
Drill row -- Thiram (50%)	6	27	126.4
Seed treated with Thiram Naugets slurry	6	--	104.7
Drill row -- Thiram (50%)	6	13	173.2

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE; AND OKLAHOMA A & M COLLEGE, STILLWATER, OKLAHOMA

COTTON SEEDLING TESTS WITH CHEMICALS USED AS PROTECTANTS AGAINST RHIZOCTONIA SOLANI IN THE GREENHOUSE

L. A. Brinkerhoff, Bill B. Brodie, and R. A. Kortsen

Seed decay and seedling infection are two important factors of a complex that contributes to poor stands of cotton in Oklahoma. After extensive surveys, Ray and McLaughlin concluded that Rhizoctonia solani Kuehn was the most important of some 14 fungi which they found attacking cotton seedlings in 21 different Oklahoma counties.[1] With these studies in mind, R. solani was selected as a test pathogen in an attempt to evaluate different chemicals as seed protectants and soil fungicides.

Our studies are presented as a progress report for consideration by others who are working on the rhizoctonia problem.

BAND TREATMENTS: Several seed treatment fungicides were compared at different rates by mixing with sand and applying in the drill row in flats of soil that had been artificially infested with R. solani. Four tests were made, and in each instance the treatments were randomized and replicated four times. The seeds were planted in furrows 1/2 inch deep and 1/2 inch wide in flats partially filled with moist soil. Each flat contained 4 to 8 rows, depending upon number of chemicals to be tested, and 20 seeds were planted per row. A single lot of the upland cotton variety Stoneville 62 was used throughout and consisted of the heavy fraction of gravity graded acid-delinted seed. Fungicides were not applied to the seed except for checks in three of the tests.

The amount of chemical for each row was mixed with 50 grams of sand and spread evenly over the seed in a band which was approximately 1/2 inch wide and 1/4 inch deep. Later, approximately 1/2 inch of non-treated sterilized soil was spread evenly over the surface of each flat.

Inoculum of a single isolate of R. solani originally obtained from cotton was grown on steamed grain sorghum seed for eight days. Hyphal suspensions were prepared by chopping the cultures in a food blender with water and then straining through several layers of cheesecloth so that the resulting suspension would pass through a sprinkling can. Hyphal suspensions were standardized by diluting the contents of each 250 ml. flask of grain sorghum inoculum with sufficient water to make 2 liters.

For the first test, 600 ml. of the diluted hyphal suspension was thoroughly mixed with the soil contained in each flat. For Tests 2 and 3, 333 mls. of inoculum was sprinkled evenly over the surface of each flat at the seed level just prior to planting. Test 4 was made in the same soil used for Test 2, but in the meantime it was remixed, dried and screened. Additional inoculum was not added. Steam sterilized soil was used for Tests 1 and 3 but not for 2 and 4.

Results. -- Data for emerged and surviving seedlings for the four tests are in Table 1. At 7.5 and 3.8 lbs. per acre, all of the chemicals were fairly effective in preventing seed decay and pre-emergence injury. At the lower rates, Arasan and Dow 9B were somewhat more effective than Spergon. Post-emergence damping-off was most severe with Phygon and Ortho 406 (captan). Ceresan M gave good protection in one test at 7.5 and 3.8 lbs. per acre, but caused injury to the roots of the seedlings. Phygon also appeared to be somewhat injurious in that the seed coat adhered and caused damage to the cotyledons. Seedlings emerged at least one day earlier when treated with Ortho 406.

In Test 2, with a high inoculum level, seed treatment (Ceresan M at 2 gms. per Kg. of seed) was not nearly so effective as band treatment. However, in Test 4, with a lower inoculum level, seed treatment (Spergon 3 gms. per Kg. of seed) was about as effective as row treatments.

COMPARISON OF TREATED AND NON-TREATED SEED AT DIFFERENT INOCULUM LEVELS: The previous series of tests indicated that the effectiveness of chemical treatments was probably related to the amount of inoculum added to the soil. Therefore the following test was made with different inoculum dosage rates to determine what effect this might have on seed treatment. The dosage rate for Tests 2 and 3 of the band treatments (333 ml. per flat) was selected as the highest level and four new rates determined by dividing this amount by 3, 9, 27,

[1] Ray, W. W. and J. H. McLaughlin. 1942. Isolation and infection tests with seed- and soil-borne cotton pathogens. Phytopath. 32: 233-238.

Table 1. Greenhouse tests in soil infested with R. solani comparing several chemicals applied as band treatments to the drill row.

Treatment	Test 1	Test 2		Test 3		Test 4[a]	
	7.5 lbs/acre[b]	7.5 lbs.	3.8 lbs.	1.97 lbs.	0.9 lbs.	1.97 lbs.	0.9 lbs.
	Emerged seedlings (% of seed planted)						
Drill row							
Spergon	62	78	71	27	15	66	53
Phygon	42	67	76	--	--	--	--
Arasan	62	80	82	69	48	71	62
Ortho 406	72	76	78	--	--	--	--
Ceresan M	--	73	76	--	33	50	52
Dow 9B	--	--	--	54	33	69	73
P-162	--	--	--	--	--	56	49
Seed treatment only							
Ceresan M	--	48		--		--	
Spergon	--	--		--		63	
No treatment							
None (check)	6	8		4		28	
	Surviving Seedlings (% of seed planted)						
Drill row							
Spergon	55	48	41	1	0	62	47
Phygon	25	40	41	--	-	--	--
Arasan	53	53	53	14	5	70	58
Ortho 406	52	34	32	--	-	--	--
Ceresan M	--	66	61	--	-	45	42
Dow 9B	--	--	--	14	2	69	70
P-162	--	--	--	--	-	54	48
Seed treatment							
Ceresan M	--	12		-			
Spergon	--	--		-		59	
No treatment							
None (check)	3	0		0		22	
L.S.D. - 1%	33	17		-		19	

[a]The soil was the same as used for Test 2; additional inoculum was not added.
[b]Equivalent to .26 gms. per linear ft., calculated on the basis of 40-inch row spacings.

and 81. The same planting procedure was followed as previously described, using steam sterilized soil. Hyphal suspensions of R. solani were prepared as previously described and sprinkled on the soil at the seed level. One 2-liter stock suspension prepared from one flask of inoculum was sufficient for the entire test. Acid-delinted "heavy" seeds were treated with Ceresan M and Spergon as dusts at 3 gms. per Kg. of seed, and similar seeds were used without treatment.

Results. -- Percentages for emerged and surviving seedlings are in Table 2. The data indicate that pre-emergence injury was definitely related to dosage rates when R. solani was applied as a hyphal suspension. The relationship seemed to hold for both treated and non-treated seeds.

Emergence was almost nil for the check at the three highest dosage rates, but was 45.0 and 63.5 percent respectively at the two lower rates. The percentage of seedlings killed was fairly comparable at all the different dosage levels.

Seed treatment offered considerable protection only at the lower dosage levels. Spergon gave considerably better protection than did Ceresan M.

Table 2. Comparison of treated and non-treated cotton seed in previously sterilized soil infested with different amounts of a hyphal suspension of R. solani.

Amount of inoculum in mls. per flat	Kind of seed treatment: None %	Ceresan M %	Spergon %	Average for inoculum dosage rates %
	Seedlings emerged after 6 days			
333.0	0.0	0.5	9.5	3.3
111.0	0.5	0.0	26.5	9.0
37.0	2.5	6.5	29.5	12.8
12.3	26.5	22.5	29.0	48.8
4.1	45.0	63.5	88.0	65.5
	Seedlings surviving after 18 days			
333.0	0.0	0.5	0.0	0.2
111.0	0.0	0.0	12.0	4.0
37.0	0.5	0.0	14.5	5.0
12.3	5.5	17.0	37.5	20.0
4.1	13.0	36.5	78.0	42.5

DRILL ROW AND SEED TREATMENT COMPARISONS: Seven tests were made in greenhouse benches in non-sterilized soil that was artificially infested with R. solani. Part of the tests were conducted at relatively cool temperatures, 65° to 75° F during the day and 60° to 65° for the nights, while temperatures for the other tests ranged between 85° to 90° during the day and 65° to 70° at night. Hyphal suspensions of inoculum were prepared as previously described and thoroughly mixed with the soil to a depth of 4 inches. Approximately 100 mls. of the standard hyphal suspension were used per square foot of bench space.

In four of the tests the chemicals were mixed with sand and applied to the open row after the seeds were planted. All of the exposed surface of the furrow was covered and then the soil filled in over the seed with a small garden rake. In three additional tests the chemicals were thoroughly mixed with all of the soil from the planting furrow by shaking soil and sand-chemical mixture together in large paper sacks. The treated soil was then placed over the seed.

In some instances the seeds were treated for the drill row treatments; in others they were not. Seed treatment only was compared with the drill row treatments in each test.

An additional test was made in sterilized soil in which Mathieson 275 (pentachloronitrobenzene) and Arasan were compared both as drill row and as seed treatments. Flats of soil were artificially infested with 50 mls. of the standard hyphal suspension of R. solani at the seed level prior to planting. The soil that covered the seed was removed and thoroughly mixed with the chemicals. After planting, the flats were held in a cold chamber at 64° F for five days and then removed to a greenhouse and held at 70° to 85°.

Results. -- Data for four of the seven tests are shown in Table 3. Seed treatment alone was almost as effective as chemicals applied to the open drill row. When the chemicals were thoroughly mixed with the soil, however, post-emergence injury was reduced.

Thiram 50, Mathieson 275, and Dithane Z-78 appeared to be about equally effective in the tests held at the higher daytime temperatures; whereas in tests held at relatively cool daytime temperatures Mathieson 275 was definitely less effective. Since the soil was not sterilized, a pathogen other than R. solani may have been responsible for the different results at the lower temperature range.

In the sterilized soil infested with R. solani (see Table 4) Arasan gave better emergence, while Mathieson 275 was somewhat more effective in controlling post-emergence injury. Treating the soil covering the seed reduced post-emergence injury.

ADDITIONAL SEED TREATMENT COMPARISONS: Further tests with seed treatment fungicides were made in a greenhouse bench in about the same manner as the tests described in the section on band treatments. Inoculum was applied to the open rows with a sprinkling can at 6 mls. of the standard hyphal suspension per linear foot before planting. Plots were single

Table 3. Cotton seedling tests in a greenhouse bench comparing chemicals applied to the drill row and to the seed in non-sterilized soil artificially infested with R. solani.

Treatment	Rate (lbs. per acre or gms. per Kg.)	Percentage of seedlings Emerged	Surviving	Free of lesions (After 3 weeks)
		Test 1 (60° to 75° F.)		
Drill row only (applied to the open row)				
Thiram 50 (50%)	4 lbs.	59.4	52.4	----
Dithane Z-78 (65%)	4 lbs.	68.0	46.0	----
Mathieson 275 (20%)	20 lbs.	42.0	31.0	----
Seed treatment only				
Arasan SF-X (sl.)	3 gms.	71.0	50.4	----
		Test 2 (65° to 90° F.)		
Seed treatment[a] + drill row (chemical thoroughly mixed with soil covering seed)				
Thiram 50 (50%)	4 lbs.	81.0	56.8	----
Seed treatment only				
Arasan SF-X (sl.)	3 gms.	61.8	25.5	----
Setrete (liquid)	3 mls.	70.0	30.5	----
Vancide 51 (liquid)	6 mls.	53.0	20.5	----
None (Check)	----	13.8	5.0	----
		Test 3 (65° to 90° F.)		
Drill row only (applied to the open row)				
Mathieson 275 (20%)	5 lbs.	42.0	28.0	----
Mathieson 275 (20%)	10 lbs.	49.0	36.0	----
Mathieson 275 (20%)	20 lbs.	52.5	41.5	----
Mathieson 275 (20%)	40 lbs.	57.5	46.5	----
Thiram 50 (50%)	4 ibs.	81.0	46.5	----
Seed treatment only				
Arasan SF-X (sl.)	3 gms.	81.5	41.0	----
Agrox (sl.)	3 gms.	87.0	59.5	----
None (check)	----	32.0	13.5	----
		Test 4 (65° to 90° F.)		
Seed treatment[a] + drill row (chemicals thoroughly mixed with soil covering seed)				
Mathieson 275 (20%)	10 lbs.	69.0	62.7	44.7
Mathieson 275 (20%)	20 lbs.	75.0	65.0	48.2
Mathieson 275 (20%)	40 lbs.	75.0	68.0	41.7
Dithane Z-78 (65%)	5 lbs.	78.7	62.3	39.6
Thiram 50 (50%)	4 lbs.	82.3	75.7	44.9
Seed treatment only				
Arasan SF-X (sl.)	3 gms.	65.0	46.7	23.6
Ceresan M (sl.)	3 gms.	73.7	47.7	23.8
Dow 9B (sl.)	3 gms.	86.0	60.3	21.5
None (check)	----	13.7	8.0	4.2

[a]The seed treatment in each instance was Arasan SF-X (sl.) at 3 gms. per Kg. of seed.

Table 4. Cotton seedling test comparing Mathieson 275 and Arasan in previously sterilized soil artificially infested with R. solani.

Treatment	Rate	Seedlings emerged %	Seedlings surviving after 3 weeks %
Seed treatment + drill row[a]			
Arasan SF-X, + Arasan (50%)	4 lbs./acre	90.2	63.5
Arasan SF-X, + Math. 275 (50%)	4 lbs./acre	71.7	60.1
Math. 275, + Math. 275 (50%)	4 lbs./acre	56.8	43.4
Seed treatment only			
Arasan SF-X (sl.)	5 gms./Kg.	85.2	11.7
Mathieson 275 (sl.)	5 gms./Kg.	71.8	38.4
None (check)	----	5.0	0.0
L.S.D. - 5%		19.5	

[a]The soil covering the seed was removed and thoroughly mixed with the chemicals. The seed treatments were applied as slurry preparations at 5 gms. per Kg. of seed.

rows 3 feet long, each planted with 100 seeds. The soil was not sterilized.

Seeds for tests 2 and 3 were obtained from Dr. C. H. Arndt of the South Carolina Agricultural Experiment Station and were from the same lots as used in regional cotton seed treatment tests. The reginned seeds (Test 3) were heavily infested with spores of the anthracnose fungus (Glomerella gossypii).

An additional test was made in which acid-delinted seed were coated with Thiram Naugets and Mathieson 275. A 3% methyl cellulose gel was used to stick 3, 9, 27, 81, and 243 gram of chemical per Kilogram of seed. Checks included untreated acid-delinted seed and Ceresan M as a seed treatment at 3 and 6 gms. per Kg. Drill-row treatments in which the soil covering the seed was thoroughly mixed with Thiram and Mathieson were also included. The test was made in sterilized soil in flats with eight different treatments per flat. Diluted hyphal suspensions of R. solani prepared as for previous tests were used at 50 ml. per flat.

Results. -- In the greenhouse bench tests, striking differences were obtained between treated and non-treated seed (see Table 5). The date indicate that seed treatment was relatively effective against seed decay at the inoculum levels used. Post-emergence injury, however, was not adequately controlled, although it was generally less in plots which had the highest emergence. Dow 9B, Arasan, and Seedox appeared to be somewhat superior to the other chemicals tested.

In the test with coated seed, emergence was relatively good for all of the treatments including the non-treated checks. Hence, it was obvious that the inoculum did not produce the usual amount of pre-emergence injury. Before the test was concluded, however, damping-off had become severe in all of the treatments except some of those involving Mathieson 275. The test is being repeated with a higher inoculum potential and wider row spacings.

Discussion and Summary

The results indicated that all of the chemicals tested gave some protection against R. solani.

The striking differences in emergence in artificially infested soil showed that seed treatments offered considerable protection against seed decay. However, with increased dosages of inoculum, seed treatments did not insure seedling emergence. The injury apparently occurred after the seeds sprouted but before the seedlings emerged. Hence, seed treatment afforded protection only when the inoculum level was relatively low.

Post-emergence injury was variable but tended to be less in plots with the highest emergence.

Table 5. Cotton seedling tests in the greenhouse comparing chemicals as seed treatments in non-sterilized soil artificially infested with R. solani.

Treatment	Rate (gms./Kg.)	Percentage of seedlings Emerged	Surviving (after 3 weeks)	Roots free of lesions (3 wks.)
		Test 1		
Fuzzy seed				
Ceresan M (dust)	3	65.6	37.5	----
Dow 9B (dust)	3	76.4	64.2	----
Spergon (slurry)	3	67.2	56.4	----
Arasan (slurry)	3	63.0	47.6	----
None -- (check)	-	1.4	0.0	----
	Mean for fuzzy	54.3	41.1	
Acid delinted seed				
Ceresan M (dust)	3	53.4	13.8	----
Dow 9B (dust)	3	72.6	50.2	----
Spergon (slurry)	3	56.8	35.0	----
Arasan (slurry)	3	71.2	54.2	----
None -- (check)	-	3.0	0.8	----
	Mean for acid delinted	51.4	30.8	
		Test 2		
Acid delinted seed				
Spergon slurry	10	36.8	32.4	8.0
Ceresan M (slurry)	2	52.8	38.8	5.6
Dow 9B (slurry)	2	61.6	56.4	16.8
Arasan SF-X (slurry)	5	64.4	52.8	15.8
Captan 406 (slurry)	5	59.6	26.4	15.6
Seedox (3%) (slurry)	15	70.8	60.8	23.6
Penetrate (liquid)	2	16.4	14.0	3.2
Vancide 51 (liquid)	10	48.0	30.8	7.6
None -- (check)	-	15.6	9.6	4.0
		Test 3		
Reginned seed				
Ceresan M slurry	2.7	64.0	24.0	2.0
Cow 9B slurry	2.7	65.2	21.2	2.4
Captan 406 slurry	5.0	46.8	7.6	0.8
Seedox (7.5%) slurry	9.0	76.6	19.2	2.4
Agrox slurry	2.1	56.4	34.0	6.0
Pentrete liquid	2.1	61.6	38.4	6.0
MEML liquid	5.0	47.6	11.6	0.4
MEMA liquid	2.4	52.0	12.8	1.2
None -- (check)		16.4	8.8	3.6
		Test 4		
Acid delinted seed				
Thiram 50	3	73.4	67.8	----
NP-1083	3	75.7	46.3	----
None -- (check)	-	51.2	39.4	----

In the earlier tests in which the soil was treated, Arasan and Thiram 50 caused the least injury to the cotton seedlings and were fairly consistent in controlling R. solani. Later, Mathieson 275 and Dithane Z-78 gave comparable results except at low temperatures (60° to 75° F.). As the soil for these tests was not sterilized a pathogen other than R. solani may have accounted for the differences obtained.

Of the chemicals tested in the soil, Mathieson 275 appeared to be the least injurious to cotton seedlings.

Two tests indicated that Mathieson 275 had a longer residual effect than did Arasan or Thiram. The apparent discrepancy between the results reported here and those recently reported by Arndt may be due to differences in methods which emphasized residual differences of the chemicals tested.[2] Arndt obtained effective control of R. solani with only Mathieson 275, Dowicide F, and Seedox.

Chemicals afforded more protection when thoroughly mixed with the soil covering the seed than when applied to the exposed surface of the open drill row. This was especially true for post-emergence damage.

The tests indicate that seed treatment probably has considerable value in protecting cotton seed from seed rot caused by R. solani.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE; AND DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OKLAHOMA A. & M. COLLEGE, STILLWATER, OKLAHOMA

[2]Arndt C. H. 1953. Evaluation of fungicides as protectants of cotton seedlings from infection by Rhizoctonia solani. Plant Disease Reporter 37: 397-400.

PHYSIOLOGIC SPECIALIZATION OF COLLETOTRICHUM HIBISCI POLL. ON KENAF

Thomas E. Summers

Abstract

Physiologic races of Colletotrichum hibisci Poll. were demonstrated, using pure line selections of kenaf, Hibiscus cannabinus L., as differentials. Three races were found to be present in Florida in 1953.

The information reported herein is a part of a general study of the disease of kenaf, Hibiscus cannabinus L., caused by Colletotrichum hibisci Poll. It has been reported by Pate[1] that certain first generation selfed lines of kenaf appeared to be highly resistant to C. hibisci. Unexplained seedling, plant, flower, and capsule infections within these lines in later generations led to the collecting and testing of different isolates of the fungus on some of the available lines of kenaf. The existence of physiologic races of C. hibisci is probably of interest to plant breeders of kenaf in other areas, as well as in Florida, and this report is being made at this time for that reason.

MATERIALS

The kenaf lines used in this study were selected from the Salvadorian variety of kenaf and had been selected in part due to their reaction to inoculum referred to in this report as isolate 3. These lines were known to be fairly uniform with regard to their reaction to this isolate. All seed were furnished by Dr. J. B. Pate of the Cotton and Other Fibers Section, Field Crops Research Branch, United States Department of Agriculture. The several fungus isolates used were made by the writer from single pustules (later transfers were made from single hyphae tips) during the year 1953, with the exception of isolate 3 which was made by Dr. E. C. Tullis from diseased kenaf from Florida in 1952.

A hypodermic needle was used to make all inoculations by pricking the plant near its growing tip through a droplet of a spore suspension of the isolate. All the inoculations were made either in the field near Lake Worth, Florida or in the greenhouse at the Everglades Experiment Station, Belle Glade, Florida.

RESULTS

Five days after inoculation, the plants were examined for evidence of infection and the number of infected plants and class reaction recorded. Since certain lines were known to become infected (develop stem lesions), yet continue to grow and develop more or less normally, a second examination was made ten days after inoculation in order to determine more accurately the degree of retardation of growth that the organism caused. The distinction between the intermediate (I) and susceptible (S) classifications listed below is based on this retardation of growth, or lack of it, following infection. This seems to be a valid characteristic since it has been observed to hold true through several generations of selecting in these lines.

The lines of kenaf used were satisfactory with regard to homozygosity and their reaction was sufficiently constant to permit the establishment of five reaction classes as follows:

1) Complete susceptibility (CS) -- extensive coalesced stem lesions and death of tip of plant.

2) Susceptible (S) -- individual stem lesions, retardation of growth, tip not killed but usually curled.

3) Intermediate (I) -- individual stem lesions, no appreciable retardation of growth.

4) Resistant (R) -- excessive callus formation or very small lesion at point of inoculation only, tip may curl slightly.

5) Highly resistant (HR) -- no reaction.

---

[1] Pate, J. B. 1953. Resistance in kenaf to Colletotrichum hibisci. Phytopath. 43: 647-648.

FIGURE 1. A -- kenaf line BG-51-3 inoculated with race 1 (left) and race 2 (right) of Colletotrichum hibisci. B -- line BG-52-18 inoculated with race 1 (left) and race 2 (right) of C. hibisci.

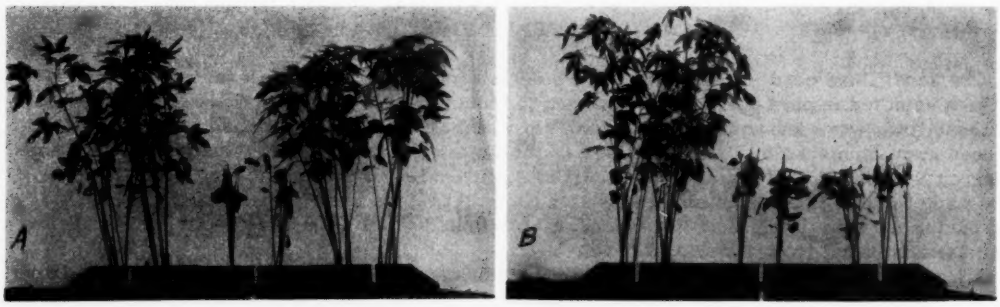


FIGURE 2. Kenaf lines, left to right, BG-52-1, BG-51-3, and BG-52-18. A -- inoculated with race 1 of Colletotrichum hibisci; and B -- with race 2. Note that BG-52-1, which has an intermediate (I) class reaction to both races, continued to grow even though it developed stem lesions.

The reaction of plants grown in the greenhouse and those grown in the field was similar for plants of comparable age. The reaction of 15 lines of kenaf, 2 introductions of Hibiscus sabdariffa, one of Hibiscus sp., and cotton (Coker 100) to three of the fungus isolates used in this study is presented as Table 1. Other isolates tested caused plants to react identically as one or another of the isolates recorded in Table 1, or the differences were so slight that until they can be tested on additional differentials they cannot be considered as separate strains or races.

The complete susceptibility (CS) reaction of kenaf BG-51-3 to isolates 1 and 2 (race 1 and race 2 of C. hibisci) is illustrated in Figure 1, A, while the highly resistant (HR) and complete susceptibility (CS) reaction of BG-52-18 to race 1 and race 2 is shown in Figure 1, B.

Figure 2, A, shows the reaction to race 1 of BG-52-1 (I), BG-51-3 (CS), and BG-52-18 (HR). Figure 2, B, shows that race 2 causes the same class reaction on BG-52-1 (I) and BG-51-3 (CS) as race 1, while BG-52-18 shows complete susceptibility (CS). Note the apparent lack of retardation of growth resulting from either race 1 or race 2 on BG-52-1, even though a considerable number of stem lesions developed from inoculation.

Owing to the small number of seed that germinated of Hibiscus sabdariffa (PI 207919 and PI 180026) and the Hibiscus sp. (PI 200834), their value as differentials cannot be evaluated at this time. It is of interest, however, that in the plants tested a difference in reaction to the different isolates was noted.

Table 1. Reaction of 15 lines of kenaf, 2 Hibiscus sabdariffa,* 1 Hibiscus sp., and 1 cotton (Coker 100) to 3 isolates of Colletotrichum hibisci.

Hosts	Isolate 1			Isolate 2			Isolate 3		
	No. plants inoculated	No. plants infected	Class reaction	No. plants inoculated	No. plants infected	Class reaction	No. plants inoculated	No. plants infected	Class reaction
Hibiscus cannabinus									
BG-51-3	10	10	CS	10	10	CS	10	10	CS
BG-52-10	24	24	CS	26	26	CS	20	20	CS
BG-52-62	11	11	S	18	18	S	14	14	S
BG-52-1	31	28	I	43	39	I	41	40	I
BG-52-6	9	7	CS	26	26	CS	14	12	I
BG-51-4	10	10	CS	30	30	CS	42	37	I
BG-52-11	16	16	I	8	8	CS	14	14	I
BG-52-18	52	0	HR	44	44	CS	50	0	HR
BG-52-28	12	0	HR	13	13	CS	20	0	HR
BG-52-33	20	0	HR	29	29	CS	33	0	HR
BG-52-34	22	0	HR	43	43	CS	23	0	HR
BG-52-37	18	0	HR	18	18	CS	23	0	HR
BG-52-61	28	0	HR	21	21	CS	23	0	HR
BG-52-66	26	0	HR	35	34	CS	9	0	HR
BG-52-78	16	0	HR	36	36	CS	13	10	R
Hibiscus sabdariffa* (PI 207919)	10	10	8-R, 2-I	12	11	I	3	2	I
Hibiscus sabdariffa* (PI 180026)	2	2	I	3	3	I	2	2	I
Hibiscus sp.* (PI 200834)	7	0	HR	38	33	I	7	7	R
Gossypium hirsutum	50	0	HR	50	0	HR	50	0	HR

*Small number due to poor germination of seed.

Table 2. Kenaf differentials and their reaction to 3 physiologic races of Collectorichum hibisci.

Differential	Reaction		
	Race 1	Race 2	Race 3
BG-51-4	CS	CS	I
BG-52-18	HR	CS	HR
BG-51-3	CS	CS	CS
BG-52-62	S	S	S
BG-52-1	I	I	I
BG-52-11	I	CS	I
BG-52-78	HR	CS	R

DISCUSSION AND CONCLUSIONS

The data from this study indicate the existence of physiologic races of Colletotrichum hibisci. BG-52-18 is highly resistant (HR) to races 1 and 3 and completely susceptible (CS) to race 2. BG-51-4 is completely susceptible (CS) to races 1 and 2 and has an intermediate (I) reaction to race 3. While the two lines mentioned above are sufficient to differentiate the now known physiologic races of C. hibisci, it appears desirable that the seven lines listed in Table 2 be used as differentials in order to attempt identification of other races of the fungus should they appear.

Race 2, as established in this report, attacked all the kenaf lines used in this study; however, the reaction of certain lines was of the intermediate (I) class, which is considered a type of resistance or tolerance. To determine if material possessing a higher type resistance to race 2 exists, this study will be extended to include the testing of all potential sources of resistance available.

The determination and establishment of physiologic races within C. hibisci will partially explain the conflicting reaction certain lines of kenaf have given when inoculated with pure cultures of the fungus and later became infected when grown under field conditions.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, BELLE GLADE, FLORIDA.

ACCELERATED SCREENING TESTS OF FUNGICIDES FOR CONTROL OF BLACK STRIPE OF THE HEVEA RUBBERTREE[1]

J. B. Carpenter[2]

Black stripe is a major disease of the tapping panel of the hevea rubbertree, Hevea brasiliensis (Willd. ex A. Juss.) Muell. Arg. and other species. It represents an important phase of the disease complex caused by Phytophthora palmivora Butler that may involve any or all of the aerial portions of the tree and root collar. The symptoms, development, and effects of the disease under Costa Rica conditions parallel its typical behavior in the principal Far Eastern rubber-growing areas (1, 9), Liberia (3), Brazil (5), and elsewhere.

Beginning in September 1950, the Phytophthora disease built up to epidemic proportions in the principal rubber plantings in the Atlantic Zone of Costa Rica (2, 6, 7). Black stripe, which up to that time had been present but not especially severe, became an important problem in commercial and experimental plantings at Cairo (footnote 3), Turrialba, and Los Diamantes. It has continued to be serious whenever circumstances favor its development.

Natural inoculation of tapping panels under conditions fostering severe disease development is potentially much better for obtaining infection than artificial inoculation. This results from the continuous presence of an abundant inoculum, conditions favoring infection, and adverse circumstances for application of control measures. For that reason, field testing is a vital step in the selection of practical fungicides. Nevertheless, such favorable conditions are of sporadic occurrence, both as to time and place, and require the tests to be carried on for perhaps one year or longer before getting definite results. This long period is too uneconomical for preliminary screening tests, especially when results are needed urgently. Therefore, methods were sought for accelerating the preliminary testing of fungicides by means of artificial inoculation of the tapping cut.

Factors that commonly limit the success of measures for control of tapping panel diseases, even with an otherwise adequate external fungicide, are (a) inoculation and infection of the tapping cut during the period between tapping and treating, (b) delayed application, (c) poor application, (d) dilution or removal of the fungicide by rains before drying, and (e) late flow, which interrupts the treated surface and opens potential infection courts.

---

[1] Cooperative investigation of the United States Department of Agriculture and the Ministry of Agriculture and Industry of Costa Rica, done at the U. S. Department of Agriculture Cooperative Rubber Plant Field Station at Turrialba and the Substation at Los Diamantes, near Guapiles, Costa Rica.

Grateful acknowledgment is made to: E. P. Imle and W. E. Manis for facilities provided at the Turrialba and Los Diamantes stations, respectively; A. V. McMullan for valuable suggestions on the inoculating technique; M. H. Langford, E. P. Imle, and A. V. McMullan for criticism of the manuscript, all of the aforementioned persons being in Field Crops Research Branch, Agricultural Research Service; J. R. Havis and K. L. Olsen, Interamerican Institute of Agricultural Sciences, Turrialba, for consultation and for expediting certain materials; and E. C. Calavan, University of California, Riverside, for information on the use of captan fungicides.

Appreciation is expressed to the following firms for excellent cooperation in providing experimental materials and information on their use: American Colloid Company, Chicago; Attapulgus Clay Company, Philadelphia; California Spray Chemical Corporation, Elizabeth, New Jersey; Carbide and Carbon Chemicals Company, New York; Corn Products Refining Company, New York; Dow Chemical Inter-American Limited, Midland, Michigan; E. I. Du Pont de Nemours and Company, Inc., Wilmington, Delaware; Ethyl Corporation, New York; General Chemical Division, Allied Chemical and Dye Corporation, New York; B. F. Goodrich Chemical Company, Cleveland; Mallinckrodt Chemical Works, New York; Monsanto Chemical Company, St. Louis; Charles Pfizer Company, Inc., New York; Standard Oil Development Company, Linden, New Jersey; R. T. Vanderbilt Company, New York; H. L. Woudhuysen and Associates, New York.

[2] Field Crops Research Branch, Agricultural Research Service, United States Department of Agriculture.

[3] Langford, M. H. 1953. "Tests on panel treatments at Speedway Estate". Unpublished résumé of detailed data taken on nearly 5000 trees in extensive fungicide trials and observations on the influence of certain cultural practices under commercial plantation conditions on the Speedway Estate of The Goodyear Rubber Plantations Company at Cairo, Costa Rica, from July 1951 to September 1953.

The screening tests reported herein represent one part of a study of measures for the control of black stripe that was begun in 1951. The other part, a study of the use of recommended and experimental fungicides and other cultural practices under plantation conditions, has been conducted by M. H. Langford[3].

PRELIMINARY STUDIES

These studies were made during the period from November 1951 to January 1953 at the rubber station in Turrialba. Methods of inoculation and a group of both proprietary and locally prepared fungicides and adjuvants were tested.

No satisfactory inoculation technique was found in this preliminary work. When the trees were inoculated immediately after tapping, and thus several hours before treatments were applied, heavy general infection occurred. This was anticipated, for the sporangia and bits of mycelium were protected by the soft coagulated latex from contact with the fungicides applied to the surface. Such a method is too prejudicial where the concept of disease control is prevention and not cure. Spray inoculation of the treated panels gave inconsistent results and was unsatisfactory, even when done after nightfall on successive days to take advantage of lower temperature, higher humidity, and low light intensity,-- all factors that may favor infection.

Failure was probably due to drying of the tapping cut during the 12- to 14-hour interval between tapping and inoculation and to rapid drying of the sprayed inoculum. Ineffectiveness of this method was further shown in 1952 by a heavy natural infection that began at one side of an artificially inoculated block. Black stripe in the area of natural infection was much more severe than elsewhere. Treatments that were holding up under artificial inoculation proved to be ineffective against natural infection.

The fungicides and adjuvants that were tested during this period and found unsatisfactory are listed below. Under other conditions, or in other formulations, some might prove useful against black stripe. The fungicides were coal-tar paint (8, page 15), Treeseal (formerly E. F. 487.2) (3), Santobrite, Dithane, lime-sulfur, wettable sulfur, miscible coal-tar disinfectant, three dinitro compounds, Puratized Agricultural Spray, three experimental chemotherapeutants, Ferronol, Fylomac No. 90, and preparations of salts of cadmium, chromium, cobalt, silver, and zinc. The adjuvants were liquid latex, wheat flour, rosin residue emulsion (Nu-Film), methylene blue, red ochre, African palm oil, lubricating oil, liquid vaseline, cup grease, bentonite clays, and corn starch derivatives.

ACCELERATED SCREENING TESTS

The accelerated screening test, as now used, was developed in August and September 1953. It was applied in four trials from September 1953 to January 1954, inclusive, at the stations in Turrialba and Los Diamantes. The essential equipment and materials needed for application of the inoculation method and the tapping system are shown in Figure 1 A.

Methods and Materials

Inoculum of Phytophthora palmivora was grown on sterile cut green beans. Abundant sporangia were produced and could be washed off with a minimum of mycelial fragments and extraneous matter. Cultures 7 to 15 days old, in pint milk bottles, were used. These bottles are cheap and rugged and can easily be transported. The suspensions of sporangia and mycelium were prepared with from two to four bottles of inoculum per liter of suspension, depending upon the age and quantity of the cultures. Cultures of four to six isolates were used to make a composite inoculum.

Tapping was done on the half-spiral, alternate-day system, mostly between the hours of 6:00 a.m. and 9:00 a.m. New tapping cuts were opened for each trial, usually alternating from one side of the tree to the other. The new cut was several inches below the previous one (Fig. 1 A). Trial I (Table 1) was made at Los Diamantes, using very low-yielding trees. Trials II, III, and IV were made at Turrialba, using mostly trees with sufficient yield to flow well. About 20 percent of these trees exhibited small areas of late flow in some part of the tapping cut and thus were especially liable to infection. The exuded latex interfered with fungicide application both initially and later, by breaking through the protective film.

None of the fungicides except Treeseal were developed for treating tapping panels. Therefore, concentrations were based, where possible, on information supplied by the manufacturer for use of the same or similar materials. The fungicides were used mostly at rates somewhat higher than those suggested for application to foliage, seed, or turf. Fungicides were made up

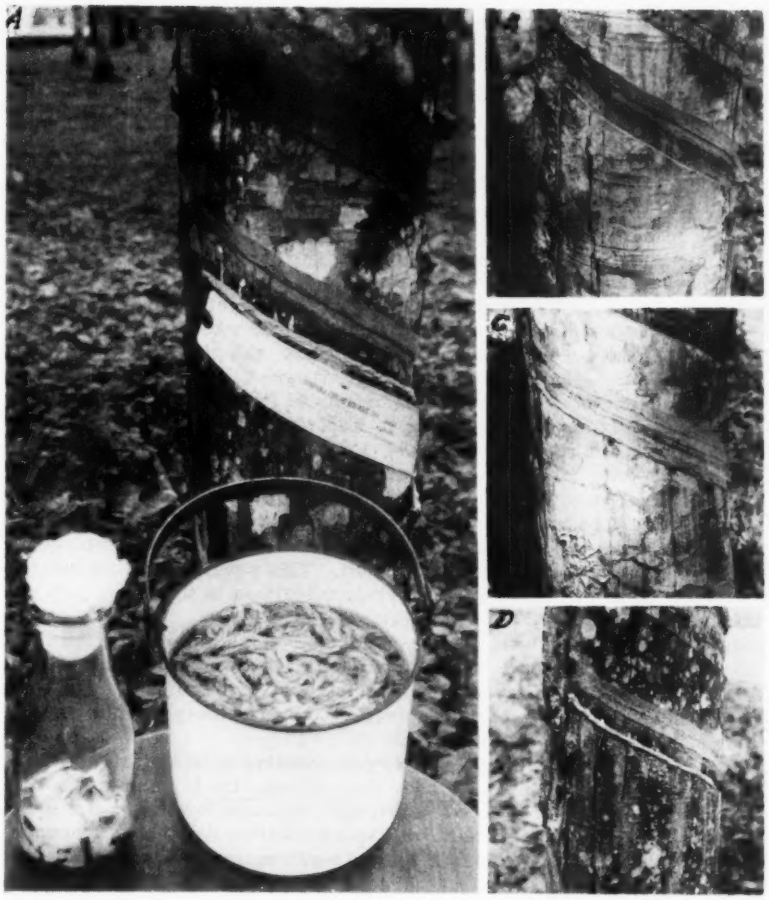


FIGURE 1. Application and results of the accelerated screening test of fungicides for control of black stripe on tapping panels of Hevea. During a 13-day period, these trees were tapped six times and inoculated 11 times. The space on the panel between tapping areas in successive trials was left to avoid invisible or latent infections from previous inoculations. Photographs were taken two weeks after the last tapping in Trial IV.

A -- Equipment needed for the method, including a culture of Phytophthora palmivora and absorbent cotton-string inoculators in a suspension of sporangia and mycelium, and position of the inoculator on the tapping cut.

B -- Untreated, uninoculated control tree with only one small infection at the upper end of the cut. The other stains are superficial.

C -- Treated with the best formulation of Orthocide 50 Wettable, inoculated, and in this instance disease-free.

D -- Treated with an unsatisfactory fungicide, inoculated, and as severely diseased as A.

once a week as water-based preparations, unless otherwise noted. This facilitated the work and imposed a stability test on them. The water-based preparations were stored in glass containers and fresh portions measured out for each day's use. These were applied with small paint brushes. Treeseal, a grease preparation, was used undiluted and applied with a finger. Application of fungicides did not begin until about noon, in order to approximate the minimum interval between tapping and treating that would occur in a commercial plantation. This is important from the standpoint of natural infection. The period of three to six or more hours during which the cut is unprotected and especially susceptible to infection is believed to be one of the principal periods during which infection occurs under naturally severe disease conditions (cf. PRELIMINARY STUDIES). Sporangia that lodge in the latex on the cut may germinate and grow into the bark, protected from external fungicidal action by the coagulated latex that remains in the cut after tapping. While attempts to study germination and growth in latex have been unsuccessful because of the opacity of latex even when diluted, there is no reason to doubt that latex offers an excellent medium for development of the fungus.

The inoculation technique was based on the combined use of string inoculators and spray inoculation. Inoculators were made of 20 cm. pieces of absorbent, twisted, cotton string about 5 mm. thick. These were boiled twice in tap water and rinsed to remove any noxious materials that might be present. The strings were dampened and dipped in groups of about 50 into a heavy suspension of sporangia and mycelium of Phytophthora. They were agitated well to insure thorough wetting and adherence of infective material and then transferred to one or more small enameled pails for field use (Fig. 1 A). By attaching the pail to a belt, thus leaving both hands free, one man can inoculate more than 300 trees per hour. Fresh inoculators were used for each tapping cycle of two days.

Inoculators were applied in mid- to late afternoon on the same day as tapping after the fungicidal treatments were applied and allowed to dry. An inoculator was placed in the upper part of each freshly treated tapping cut to be tested. This provided a fixed location for successive inoculations, with the possibility of further infections in the lower part of the cut by drainage of infective material from the inoculator.

Spray inoculations were made on the alternate, non-tapping days, for the inoculators could not be replaced with fresh ones without frequently stripping off both the coagulum and the fungicide. A brief burst of spray was applied to each inoculator and tapping cut, using a 2-gallon knapsack sprayer reserved for this use. About 125 nine-year-old trees were inoculated with 1 liter of inoculum.

The most severe inoculation was obtained by inoculating on four or six consecutive days in each of two or three successive weeks. Under this system, the tapping cuts were subjected to the inoculum for periods of about 40 hours between taps and for two or three tapping cycles each week. Although the first infections were apparent within three days on control trees or those with ineffective fungicides, prolonged testing was required to get a rapid and severe involvement of the tapping cut.

The results of inoculation were scored on the basis of the 20 cm. long section of the tapping cut immediately beneath the inoculator. Infections were often sharply limited to this area. The four classes of infection were: A, no infection; B, 0 to 12.5 percent infection, or no more than a total of 2.5 linear cm. of lesions on the cut; C, 12.5 to 50 percent infection, or no more than a total of 10 linear cm.; D, 50 to 100 percent infection, or possible involvement of the entire 20-cm. length. The secondary category, B, consisted mostly of scattered black stripes and rarely solid areas. It would not ordinarily enter into plantation or commercial evaluation of the disease, since many of these small lesions never develop any further. Therefore, in the tabular presentation of results, Classes C and D (Table 1) are considered as the important categories of disease from a commercial viewpoint.

In order to verify the effectiveness of the inoculation technique, in each trial two sets of control trees were used. One set was inoculated artificially and the other left to receive natural infection.

Results

The inoculation technique was successful, as indicated by (a) the amounts of disease recorded in Table 1 for the artificially inoculated and naturally infected control trees (Fig. 1 A and B), and (b) by the elimination of 19 of the 21 fungicides at the concentrations and in the formulations used. Phenyl mercury acetate was fungicidal but too phytotoxic for further use.

Orthocide 50 Wettable, a captan fungicide (4), California Spray Chemical Corporation, was the only fungicide that gave sufficient control (Fig. 1 C) to warrant repeated trials (Table 1). In Trial I, Orthocide with Good-rite p.e.p.s., B. F. Goodrich Company, gave

Table 1. Comparative results of accelerated screening tests for fungicides to control black stripe on tapping panels of the Hevea rubbertree. The data for inoculated and natural-control trees and for the most promising fungicides are given.

Treatment				Trial number[a]	Total trees No.	Number of trees infected arranged by classes of infection[b]				Proportion of trees infected (percent)	
Fungicide	%	Adjuvant	%			A	B	C	D	Total	Classes C and D[c]
Orthocide	2.0	None		III	15	7	3	3	3	60	40
	2.0	Filmfast	1.0	III	14	7	5	0	2	50	14
	2.0		1.0	IV	18	4	10	4	0	78	22
	2.0		0.5	IV	17	9	5	3	0	47	18
	1.5		1.0	IV	18	6	8	3	1	67	22
	1.5		0.5	IV	18	6	9	3	0	67	17
	1.0		1.0	IV	18	7	5	6	0	61	33
	1.0		0.5	IV	18	2	7	6	3	89	50
	2.0	P.e.p.s.[e]	1.0	I	40	37	3	0	0	8	0
	2.0		1.0	III	15	0	2	4	9	100	87[e]
	2.0		1.0	IV	18	3	8	5	2	83	39
	2.0		0.5	IV	18	4	8	1	5	78	33
	1.0		1.0	I	40	35	4	1	0	13	3
	1.0		1.0	III	15	0	2	6	7	100	87[e]
Treeseal[f]				I	39	7	12	7	13	82	51
				II	12	0	1	8	3	100	92
Control, natural infection				I	40	33	2	0	5	18	13
				III	12	6	2	1	3	50	33
				III	15	6	5	1	3	60	27
				IV	18	10	2	2	4	44	33
Control, inoculated				I	71	0	1	7	63	100	99
				II	12	0	0	0	12	100	100
				III	15	0	0	0	15	100	100
				IV	18	0	0	0	18	100	100

[a] Trial I, Los Diamantes, 4 replications of each treatment except inoculated control which had 8, inoculated 12 out of 29 days. Trial II, Turrialba, 3 replications, inoculated 7 out of 13 days. Trial III, Turrialba, 3 replications, inoculated 10 out of 22 days. Trial IV, Turrialba, 3 replications, inoculated 11 out of 13 days.

[b] Classification based on percent linear extension, determined on 20-cm. long area immediately beneath the inoculator. Class A, no linear extension; Class B, 0-12.5 percent linear extension; Class C, 12.5-50 percent linear extension; and Class D, 50-100 percent linear extension.

[c] Classes C and D give a truer picture of fungicidal behavior, as Class B would not ordinarily be included in a plantation rating of disease.

[d] Orthocide 50 Wettable.

[e] P.e.p.s. = Good rite p.e.p.s. It is belived that the lot used in Trial III had deteriorated in storage. The fresh lot used in Trial IV gave better results.

[f] Treeseal included as a recommended fungicide for control of black stirpe (3).

outstanding control. In Trials III and IV, the degree of control was more in line with what might be expected of a suitable fungicide. Although no uninoculated trees were treated with Orthocide, in view of its behavior under heavy inoculation, good control would undoubtedly have resulted. On the basis of these limited results, field trials of Orthocide have been started at the Substation in Los Diamantes on a total of about 2000 trees and will continue at least until the end of 1954.

Treeseal was used as a standard of comparison in Trials I and II, as it has been recommended for use against black stripe (3). It did not give adequate control. Additional information on this fungicide was obtained at the Substation in Los Diamantes. A block of 54 experimental clones was examined five months after being brought into tap on the half-spiral, alternate-day system. Many of these clones, of both Hevea brasiliensis and H. brasiliensis x H. benthamiana Muell. Arg. origin, are very susceptible to Phytophthora. Treeseal was applied after every tap, either on the same or the following day. Only infected trees with more than 1 sq. in. (6 sq. cm.) of diseased bark were counted, in conformance with the system used by Langford (footnote 3). The incidence of infection for each category of clones was as follows: (a) known control clones, GA 49 and GV 37, based on 21 uniformly spaced lines representing one-third of the total number of controls, 16 percent of 91 trees; (b) 54 experimental clones, 35 percent of 495 trees. Of these experimental clones, 27 each had more than one-half of the trees infected, or 84 percent of 179 trees, and 17 each had 100 percent infection on a total of 88 trees.

Since control of panel diseases constitutes an important part of the program in other rubber research centers, it may be profitable to list the other fungicides tested. Under different circumstances, some might be useful. In the following list, the active ingredient and the maximum concentration used, expressed as percent of active ingredient, are shown for each fungicide. The minimum percentage of diseased trees in classes C and D (cf. Table 1) is underscored. The source of each fungicide is given in abbreviated form (see footnote 1). Good-rite p.e.p.s., B. F. Goodrich Company, and Filmfast, General Chemical Division, were used as stickers at 1.0% and 0.5%, respectively, and are designated as P and F.

B 622, 2,4,-dichloro-6-(-cloroanilino-s-triazine), 0.5, + F, 100. Ethyl Corp.
B 856, composition unknown, 0.5, + F, 100. Ethyl Corp.
CADMINATE, Cd succinate, 0.25, + P, 86. Mallinckrodt.
CADMINE, Cd ricinoleate, 0.5, 85. Woudhuysen.
CALO-CLOR, HgCl and $HgCl_2$, 0.25, + P, 100. Mallinckrodt.
CERESAN M, ethyl Hg p-toluene sulfonanilide, 0.1, + P, 90. Du Pont.
COMPOUND 1189, composition unknown, 0.5, + F, 100. General Chemical.
CRAG FRUIT FUNGICIDE 341, 2-heptadecyl glioxalidine acetate, 0.75, + 1.25 spray lime, + P, 80. Carbide and Carbon.
CRAG FUNGICIDE 974, 3, 5,-dimethyltetrahydrothiodiazine-2-thione, 0.5, + F, 100. Carbide and Carbon.
MALACHITE GREEN, 0.05, 100.
MELSAN, ethyl Hg phosphate, Na pentachlorophenate, Na salts of other chlorophenols, 0.25, + P, 51. Du Pont.
MERCADMINE, phenyl Hg salicylate, Cd ricinoleate, 0.05, 95. Woudhuysen.
MERCULINE, phenyl Hg salicylate, 0.025, 94. Woudhuysen.
PARAFORMALDEHYDE, 0.5, + F, 100. Du Pont.
PHENYL MERCURY ACETATE, strongly fungicidal but with too narrow limits between effective control, 0.2 percent, and phytotoxicity, 0.25 percent. General Chemical.
PHENYL MERCURY DIETHYL DITHIOCARBAMATE, 1.0, +F, 1.0, 100. General Chemical.
THIOLUTIN, 0.012, in talc and Filmfast paste, 100. Charles Pfizer.
VANCIDE 50 P.E.P.S., as the undiluted paste, gave good control when permitted to dry thoroughly. However, during wet weather this material would often be washed off before drying. Vanderbilt.
VANSEAL WAX, undiluted, 45. Vanderbilt.

Filmfast was the most satisfactory sticker used with Orthocide 50 Wettable. Good-rite p.e.p.s., another synthetic latex, Latex X-120, Monsanto Chemical Company, and an experimental sticker, RDA 156, Standard Oil Development Company, were less suitable in these tests.

SUMMARY

An accelerated screening test of fungicides for control of the black stripe disease on tapping panels of the hevea rubbertree is described. The method is based on heavy artificial inoculation of the tapping cut, following treatment with fungicides, by means of an absorbent cotton-string inoculator moistened with a suspension of sporangia and mycelium of Phytophthora palmivora. A single preliminary trial may be completed in less than one month.

Among the 22 fungicides subjected to one or more trials under this method, only Orthocide 50 Wettable has shown sufficient promise for further tests under plantation conditions. Filmfast was the most satisfactory sticker tested.

Literature Cited

1. Bugnicourt, F. 1946. La maladie des "Raies Noires" de l'écorce saignée. Cahiers I. R. C. I. [Paris] 2:41-67. [L'Institut de Recherches sur le Caoutchouc en Indochine].
2. Carpenter, J. B. 1954. An epidemic of Phytophthora leaf fall on hevea rubber trees in Costa Rica. (in press).
3. Darley, E. F., and S. B. Silverborg. 1952. Black thread of Hevea brasiliensis in Liberia. Phytopath. 42: 547-558.
4. Interdepartmental Committee on Pest Control. 1953. Captan -- a coined name for the fungicidal chemical n-trichloromethylthio tetrahydrophthalimide. Phytopath. 43: 58.
5. Langford, M. H. 1953. Hevea diseases of the Amazon Valley. Inst. Agron. do Norte (Belem, Para, Brazil), Bol. Técn. 27, 29 pp. (Summaries in English, Portuguese, French, and German).
6. Langford, M. H., J. B. Carpenter, W. E. Manis, A. M. Gorenz, and E. P. Imle. 1954. Hevea diseases of the Western Hemisphere. Plant Dis. Reptr., Suppl. 225, pp. 37-41.
7. Manis, W. E. 1954. Phytophthora leaf fall and dieback. Plant Dis. Reptr., Suppl. 225, pp. 49-52.
8. Martin, W. J. 1949. Moldy rot of tapping panels of hevea rubbertrees. U. S. Dept. Agri. Circ. 798. 23 pp.
9. Sharples, A. 1936. Diseases and pests of the rubber tree. MacMillan and Company Limited. 480 pp.

UNITED STATES DEPARTMENT OF AGRICULTURE, COOPERATIVE RUBBER PLANT FIELD STATION, TURRIALBA, COSTA RICA

A SECONDARY LEAF SPOT OF THE HEVEARUBBERTREE CAUSED BY GLOMERELLA CINGULATA[1]

J. B. Carpenter and John A. Stevenson[2]

A secondary leaf spot disease of the hevea rubbertree, Hevea brasiliensis (Willd. ex A. Juss.) Muell. Arg. and other species, caused by Glomerella cingulata (Stonem.) Spauld. & Schrenk, is described. The laboratory and field studies were conducted in Costa Rica mostly during the period from November 1950 to March 1951, and the taxonomic studies were pursued in The National Fungus Collections of the Plant Industry Station, Beltsville, Maryland.

Glomerella cingulata (syn. Neozimmermannia elasticae Koord.) has been reported as the perfect state of various species of Colletotrichum and Gloeosporium found on hevea rubber (1, 4, 6, 10, 11, 17, 18). Saccas (14) has recently discussed and illustrated Glomerella on Hevea, referring the form with which he worked to G. cingulata var. aleuritidis Saccas & Drouillon. The conidial form, however, here designated as Colletotrichum gloeosporioides Penz., is the one in which the fungus is most commonly found in nursery and field plantings. Martin (10) and Langford (6) have reported the occurrence of this species on rubber in Mexico and Brazil, respectively. There have been many studies of it other than as a pathogen of Hevea, some of the more important of which are those of Shear and Wood (16), Burger (3), Muller (12), Baker, Crowdy and McKee (2), and Leibovit (9). The fungus used in the present study appears to be the same as that studied by these authors.

Glomerella in its imperfect stage as Colletotrichum or Gloeosporium has been reported from practically all Hevea growing countries, including Ceylon, Fiji, India, Indo-China, Indonesia, Malaya, the Philippines, Belgian Congo, French Equatorial Africa, Nigeria, Sierra Leone, Uganda, and Brazil. Named species in Colletotrichum reported on Hevea are C. heveae Petch, C. derridis van Hoof (5), and C. ficus Koord. In Gloeosporium, Hevea-inhabiting species noted are G. albo-rubrum Petch, G. brunneum Petch, G. elasticae Cke. & Mass., and G. heveae Petch. From a study of the several descriptions given for these species, it does not appear that any of them can be maintained as distinct from C. gloeosporioides. In fact, this conclusion has already been arrived at for most of these so-called species by previous workers. Butler (4) considers G. elasticae and C. ficus as pertaining to the conidial stage of G. cingulata, a decision concurred in by later workers (1, 15, 18). Steinmann (17), La Rue and Bartlett (8), Sharples (15), and others have similarly placed the remaining names.

Distribution and Description

In Costa Rica, this disease has been observed at the United States Department of Agriculture Cooperative Rubber Plant Field Station at Turrialba, the Substation at Los Diamantes, near Guapiles, and the Speedway Estate of the Goodyear Rubber Plantations Company of Costa Rica at Cairo.

The disease produced by Glomerella is characterized herein as a secondary leaf spot, for that is its typical mode of development. The term anthracnose, cf. Butler (4), has not been applied to this disease as it usually connotes a disease arising typically from direct infection by a stronger pathogen. Infection by this fungus seems always to follow some adverse condition for the host. Secondary leaf spot is found most commonly in nurseries following primary infection by Dothidella ulei P. Henn., the pathogen causing South American leaf blight. The leaf spot also appears independently of leaf blight as a result of: (a) mechanical injury to the leaves, (b) sunscald or tip burn that sometimes develops on tender leaves when a prolonged period of cloudy, wet weather is followed immediately by bright, hot days, and (c) probably direct infection under the conditions referred to in b, resulting in lesions that are often apical, perhaps because water remains longer on the leaf tips.

[1]Cooperative investigation of the United States Department of Agriculture and the Ministry of Agriculture and the Ministry of Agriculture and Industry of Costa Rica.

[2]Field Crops Research Branch and Horticultural Crops Research Branch, respectively, Agricultural Research Service, U. S. Department of Agriculture.

Grateful acknowledgment is made to E. P. Imle and M. H. Langford for criticism of the manuscript.

The lesions are roughly circular to semicircular in outline, depending upon their age and location on the leaf. Single or multiple lesions may occupy over one-third of the leaf surface in severe cases of infection. Active lesions are bordered by a narrow band of translucent tissue about 1 mm. wide. Otherwise, the margin of the lesion is usually a bright brown color, fading toward the older part of the lesion into shades from tan to ashy gray. The acervuli are borne on both sides of the leaf but predominate on the ventral surface where, under favorable conditions, they produce coral pink to whitish masses of conidia. Dark brown setae are frequently, though not always, present. In younger lesions acervuli are often arranged in concentric lines, but, as the lesions enlarge, this arrangement is interrupted into short linear groups or even individual acervuli. The lesions are invaded rapidly by saprophytic fungi and these may be the conspicuous elements in the older necrotic tissues. However, when isolations are made from only the narrow margin, preferably of an active lesion, pure cultures of Glomerella may be obtained.

Mycological Studies

Perithecia were found in some routine plantings made during a preliminary examination of this leaf spot. Taking advantage of this occurrence, a study was made of 16 monospore isolates derived from three typical mass isolates. Single germinated spores on agar were transferred with a glass needle to individual agar plates and their singleness verified microscopically. Whether the spores were conidia or ascospores could not be verified with the dissecting microscope that was used. These isolates were grown on both potato-dextrose agar (PDA) and rubber-wood sticks, in order to study the different modes of sporulation and growth and to provide inoculum for infection experiments.

On PDA the ascocarps were clustered into hard, slightly erumpent, blackish groups, with the bulk of each group embedded in the agar. The individual ascocarps were globose instead of pyriform. On rubber-wood sticks, however, the perithecia developed superficially and many, single or in small groups, were distinctly pyriform.

The perithecia were identified as those of Glomerella cingulata and occurred in varying abundance in 13 of the 16 isolates cultured on PDA. Eight of these were derived from a single mass isolate that produced perithecia almost exclusively. Three of the monospore isolates produced so many acervuli and conidia that they were pinkish instead of dark gray in color, but few, if any, perithecia were formed. The data indicate that the isolates studied are homothallic. This conforms with the findings of other investigators, as reviewed by Olive (13).

Conidia of two types were common in cultures on PDA. In young cultures, or at the advancing margin of older ones, great numbers of conidia were formed directly on undifferentiated hyphae. These were more variable in length than conidia produced in acervuli and were somewhat tapered. Their maximum diameter was about equal to that of the acervular type. After the cultures were a few days old, dark-walled acervuli were produced in more or less abundance. The spores in mass were coral pink and quite uniform in size. Occasional dark brown setae were found accompanying these acervuli. On rubber-wood sticks, especially, the acervuli appeared more like dark-walled pycnidia and were either clustered or solitary.

Measurements were made of ascospores, acervular conidia, and hyphal conidia (Table 1). The dimensions of all the spore types compare favorably with the dimensions for isolates of Glomerella cingulata (16). The consistent measurements of acervular conidia from three distinct sources are interesting, in view of the variability so often encountered in studies of the asexual forms of this species.

Infection Studies

Each of the 16 monospore isolates was used in infection studies to determine the pathogenicity of this fungus. Seedling plants, from leaf-blight-resistant parents, were used to reduce complications arising from infections by Dothidella. All inoculations were made on young pendent leaflets.[3]

[3]The leaves of the hevea rubber tree are digitately trifoliate. The young leaf blades are reflexed within a few days after separation from the growing point and remain pendent until fully expanded, afterwards becoming reclinate. The leaflets are so disposed that each bounds one side of a triangular interspace with the ventral surfaces facing outwards.

Table 1. Measurements of ascospores and conidia of monospore isolates of Glomerella cingulata derived from infections on Hevea rubber leaves.

Description of culture	Number of cultures[a]	Spore size in microns					
		Maximum[b]		Minimum[b]		Average	
		Length	Width	Length	Width	Length	Width
Ascospores from 6-week-old cultures on rubber wood	6	19.3	6.1	12.3	3.5	16.2	4.8
Acervular conidia from 1-month-old herbarium specimens on leaves	4[c]	17.5	6.1	10.5	3.5	14.3	4.6
Acervular conidia from 6-week-old cultures on rubber wood	4	17.5	6.1	11.4	3.5	14.4	5.2
Acervular conidia from 6-day-old cultures on potato-dextrose agar	3	17.5	6.9	12.3	4.4	14.7	5.3
Hyphal conidia from margin of 6-day-old culture on potato-dextrose agar	4	35.0	6.9	11.4	3.5	16.2	4.8

[a]Twenty-five spores were measured for each culture; therefore, these represent 150, 100, 100, 75, and 100 spores, in descending order.
[b]These figures represent the range in size for the group and not for individual spores.
[c]The same monospore isolates were used for all measurements of conidia. In one instance only three of the isolates were available.

The inocula for these experiments were examined microscopically to be sure that they were satisfactory. Conidia germinated in four to six hours in water or on agar.

Wound inoculations were made by perforating a pair of young pendent leaflets with a lance and inserting between them a bit of cotton soaked in a suspension of spores and mycelium. The leaves and inoculum were clamped loosely between 1-inch strips of index card. Three pairs of leaflets were used for each isolate in each series of tests made in the field and lath house; six pairs of leaflets were used for the controls, which were inoculated with sterile water. After four days, the strips were removed and readings begun. The lath house tests were made with potted seedlings; those in the field employed seedling trees in nursery beds.

Inoculations without wounding were made on potted seedlings that were grown in full daylight and had normal foliage. They were protected from dew and rain by moving them into an open corridor every night and during the daytime when necessary. In this test, four leaves were used for the controls which were inoculated with sterile water. Two young, pendent leaves were inoculated with each isolate of Glomerella. The leaves were wet thoroughly with a water suspension of spores and mycelium from cultures on PDA, and a bit of cotton moistened with the same suspension placed loosely among the leaflets. Each leaf was slipped carefully into a large test tube, 37 x 195 mm., supported on a stake. The tube was lightly closed with cotton. A pad of moist cotton in the base of each tube helped to maintain high humidity. These plants were kept in light, moving shade under trees or in strong diffuse daylight. The tubes were removed after five days and the plants returned to full sun on the sixth day.

The results of the inoculation studies are shown in Table 2. The earliest visible infections occurred within 3 days following inoculation of wounded leaves. Pink spore masses were present on many lesions by the fifth day. The final readings in Tests A and B were made 7 and 12 days, respectively, after inoculation. In the series of inoculations without wounding, most of the lesions had already appeared as small flecks when the tubes were removed. When final readings were made on the twelfth day, these flecks had developed into small but definite lesions.

Table 2. Results of inoculation experiments on Hevea rubber leaves with monospore isolates of Glomerella cingulata.

Culture letter	Wound inoculations[a]			Inoculations without wounding[b]
	Test A		Test B	
	Lath house	Field	Lath house	
A	3[c]	2[d]	2[c]	2[e]
B	2	2	3	1
C	3	3	3	1
D	2	2	3	1
E	2	3	3	2
F	3	3	3	1
G	1	0	3	0
H	3	0	3	1
I	2	1	3	2
J	3	0	3	2
K	3	3	3	2
L	3	3	3	1
M	1	1	3	2
N	0	1	2	2
O	0	2	3	1
P	2	3	3	2
Control	0	0	0	0

[a]Three pairs of leaflets were used for each set of inoculations in each series. Positive inoculations are listed by pairs of leaves.
[b]Two leaves (3 leaflets each) were used. Positive inoculations listed by whole leaves.
[c]Potted seedlings apparently free of natural infections. In all cases, the lesions were associated with the artificial wounds.
[d]Nursery seedlings. The identity of the fungus was verified by reisolation from each lesion, since the risk of natural infection was greater than in the lath house.
[e]Potted seedlings. The lesions were mostly less than 2 mm. in diameter and each was verified by reisolation of the fungus.

All of the lesions resulting from wound inoculations in Test A, Field, (Table 2) and from inoculations without wounding were verified by reisolating the pathogen.

The occurrence of direct infection without wounding indicates that Colletotrichum gloeosporioides is capable of true parasitic development on Hevea. Such infections have been presumed in previous observations when there was no evidence of primary injury, other than that induced by prolonged periods of wet, cloudy weather.

The characteristic behavior of this fungus as a secondary invader also confirms its well-developed parasitism. Once established, it frequently makes an aggressive invasion of the leaf tissues. It has been found that the secondary involvement of leaf blight lesions may begin even when these are only a few days old, actively sporulating, and with no visible necrotic tissue. During attempts to isolate Dothidella from such lesions, almost equal numbers of colonies of Glomerella and Fusarium were obtained in a large number of platings. There were very few sterile leaf pieces.

Disease Control

Prior to 1951, when preparations of zinc ethylene bisdithiocarbamate (Dithane Z-78, Parzate) first came into use for nursery spraying (7), the insoluble copper fungicides gave imperfect control of leaf blight in local nurseries. As a result, many pin-point lesions of leaf blight were initiated. Even though their development was arrested by subsequent spray applications, secondary infection by Glomerella occurred in a large number of such lesions. Once established, it seemed to spread with impunity and led to serious reduction of the effective leaf surface.

Observation of the fungicide trials of Langford and Echeverri (7) showed that the progressive reduction of leaf blight damage was accompanied by a concurrent reduction in

secondary leaf spot. In the plots treated with Parzate or Dithane, there was a low incidence of both leaf blight and secondary leaf spot. With improved fungicidal control of leaf blight, Glomerella infections have become of minor importance in sprayed nurseries.

Secondary leaf spot is still, at times, a serious nuisance in nurseries planted with seed derived from leaf-blight-resistant trees. These seedlings have enough leaf-blight resistance, as a population, to grow without being sprayed. Nevertheless, some leaf blight lesions do occur and these serve as infection courts for Glomerella, which then develops typically.

Summary

A secondary leaf spot disease of the Hevea rubbertree in Costa Rica is described. The perfect stage of the causal agent is Glomerella cingulata (Stonem.) Spauld. & Schrenk; the imperfect stage is Colletotrichum gloeosporioides Penz. The perfect stage was verified by a study of 16 monospore isolates. The occurrence of perithecia in 13 of the 16 isolates when grown on potato-dextrose agar indicates homothallism in this group of isolates. Both acervular and hyphal conidia were produced in culture. Measurements of ascospores and conidia are in agreement with those previously reported for both stages of the fungus.

Inoculation studies with the 16 isolates have confirmed the observation that the fungus is primarily a secondary invader, although it may infect directly under especially favorable conditions of prolonged wet, cloudy weather.

Control of secondary leaf spot in nurseries is correlated primarily with the control of South American leaf blight, since the lesions caused by Dothidella ulei, even though arrested by later applications of spray, serve as infection courts for Glomerella cingulata. Improved control of leaf blight by preparations of zinc ethylene bisdithiocarbamate (Dithane Z-78, Parzate) has reduced this leaf spot to a factor of minor importance in sprayed nurseries. The disease continues to be a nuisance in unsprayed nurseries planted with seed from leaf-blight-resistant trees.

Literature Cited

1. Altson, R. A. 1950. Pathological Division. Report for the year 1948. In: Rubber Research Institute of Malaya. Report for the period September 1945 to December 1948. pp. 135-190. Kuala Lumpur.
2. Baker, R. E. D., S. H. Crowdy, and R. K. McKee. 1940. A review of latent infections caused by Colletotrichum gloeosporioides and allied fungi. Trop. Agr. (Trinidad) 17: 128-132.
3. Burger, O. F. 1920. Variations in Colletotrichum gloeosporioides. Jour. Agric. Res. 20: 723-736.
4. Butler, E. J. 1918. Rubber. Anthracnose. In: Fungi and disease in plants. pp. 512-515. Thacker, Spink & Company. Calcutta and Simla.
5. John, K. P. 1952. A hitherto undescribed leaf disease of hevea rubber caused by a species of Colletotrichum. Rubber Res. Inst. Malaya. Jour. 14 (278): 11-19.
6. Langford, M. H. 1953. Hevea diseases of the Amazon valley. Inst. Agron. do Norte (Belém, Pará, Brazil), Bol. Tech. 27, 29 pp. (Summary in English, Portuguese, French, and German).
7. _____ and H. Echeverri. 1953. Control of South American leaf blight by use of a new fungicide. Turrialba (Costa Rica) 3: 102-105.
8. La Rue, C. D. and H. H. Bartlett, 1923. A leaf-fall disease of Hevea brasiliensis Muell.-Arg. due to Gloeosporium albo-rubrum Petch. Papers Mich. Acad. Sci. Arts, and Letters. 2: 73-90.
9. Leibovit, A. B. 1951. Seedbed anthracnose (Colletotrichum gloeosporioides Penz. cacao strain) of cacao (Theobroma cacao L.) in Costa Rica. Unpublished thesis, 112 pp. Interamerican Institute of Agricultural Sciences, Turrialba, C. R.
10. Martin, W. J. 1947. Diseases of the hevea rubbertree in Mexico 1943-1946. Plant Dis. Reptr. 31: 155-158.
11. Mitra, M. and P. R. Mehta. 1938. Some leaf diseases of Hevea brasiliensis new to India. Ind. Jour. Agr. Sci. 8: 185-188.
12. Muller, H. R. A. 1939. Over het epidemisch optreden van de Gloeosporium-bladziekte bij Djeroek in Oost-Java. Landbouw 15: 324-345.
13. Olive, L. S. 1951. Homothallism and heterothallism in Glomerella. New York Acad. Sci. Trans. Ser. II, 13: 238-242.

14. Saccas, A. M. 1953. Les principales maladies cryptogamiques de l'Hevea en A. E. F. L'Agron. Trop. 8 (no. 2): 1-79.
15. Sharples, A. 1936. Appendix. List of fungi recorded on rubber trees in Malaya. In: Diseases and pests of the rubber tree. pp. 465-470. MacMillan and Company Limited.
16. Shear, C. L., and A. K. Wood. 1913. Studies of fungous parasites belonging to the genus Glomerella. U. S. Dept. Agr., Bur. Pl. Ind. Bul. 252, 110 pp.
17. Steinmann, A. 1922. Over de instervingsziekte bij Hevea brasiliensis. An apparently non parasitic dieback of Hevea brasiliensis. Arch. v. Rubbercult. Nederl.-Indië 6: 95-112. (English summary.)
18. Weir, J. R. 1926. A pathological survey of the Para rubber tree (Hevea brasiliensis) in the Amazon valley. U. S. Dept. Agr., Dept. Bul. 1380, 130 pp.

UNITED STATES DEPARTMENT OF AGRICULTURE, COOPERATIVE RUBBER PLANT FIELD STATION, TURRIALBA, COSTA RICA; AND THE PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION, PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND.

FUSARIUM ROOT ROT OF GUAYULE

Don C. Norton

Abstract

A root rot of guayule, caused by Fusarium solani is described. In the field, all or only part of the branches wilt. A brown to reddish-brown discoloration of the internal root tissues is evident. Inoculation tests and field observations indicate that the pathogen is not highly virulent under customary growing conditions.

During recent years, a root rot of guayule, in which isolates of a Fusarium sp. were consistently obtained, has been found during the cooler and wetter months of the year.

Fusarium spp. have been isolated previously from diseased guayule but have been regarded either as secondary invaders (2) or as saprophytic or of questionable pathogenicity (3). In 1946, Campbell and Presley (1) suggested that Fusarium spp. may cause disease under certain conditions. Descriptions of the lesions were not given in any of the previous papers.

FIGURE 1. Wilting of a guayule plant of variety 593 three weeks after inoculation with Fusarium solani. Check at right.

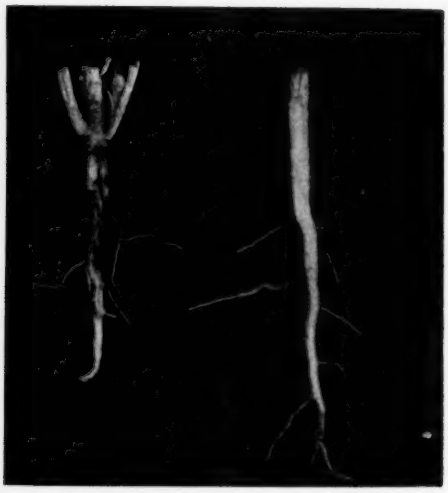


FIGURE 2. Internal discoloration of a guayule plant of variety 593 three weeks after inoculation with Fusarium solani.

In November 1951, a wilting of plants occurred in a roughly circular area in a dense eight-month-old nursery planting in southwest Texas. Examination of the internal root tissues revealed a general brown discoloration. Unless decay was in an advanced stage, the margin of the discolored area was usually very distinct from that of the healthy tissue. A Fusarium sp. was isolated consistently from the advancing edge of the discolored area. During 1952 and 1953, a similar root rot appeared on two-year-old non-irrigated plants. In some cases, the entire plant was killed while, in others, only one side seemed to be affected. The disease has not been noticed to occur in circular patches except in the nursery. Isolates of Fusarium from diseased tissue appeared to be identical with those obtained previously. The fungus has been identified as Fusarium solani Mart. emend. Snyder & Hansen.

Inoculation tests were made on guayule varieties 593 and 9800 in the greenhouse. The fungus in question was grown on sterilized oats for ten days. One tablespoonful of this mixture was buried against the tap roots of 15 five-month-old plants of variety 593 and 10 plants, the same age, of variety 9800 growing in natural unsterilized soil. As checks, the same quantity of sterilized oats was buried against the tap root of five plants of each variety. Wilting, during the heat of the day, was noted in two of the inoculated plants in approximately two weeks. During the next few days, the wilting became more severe and extended over a longer period. At the end of three weeks the plants did not recover during the cooler night temperatures (Fig. 1). When the roots were split lengthwise, a brown to reddish-brown discoloration of the internal tissues was noted (Fig. 2). The fungus was reisolated from the diseased tissue. Other inoculated plants were affected similarly but the symptoms were slower in appearing. One month after inoculation, seven plants of each variety showed no wilting. When the roots of these were split lengthwise, three of variety 593 and two of variety 9800 had a slight discoloration of the outer cortex while the remaining plants appeared entirely healthy. None of the check plants showed any external or internal symptoms of the disease.

To date, the disease has been only of minor importance in the field but has occurred often enough to bear watching. Whether this is because the fungus has not yet become established in guayule fields or because it is a weak pathogen and is destructive only in the presence of certain predisposing factors is not known. However, the latter is suspected, since only one side of some plants was affected in the field, and since disease symptoms did not develop in many of the plants in the greenhouse in spite of the high inoculum potential. Wounds caused by insects were noticed on some of the roots of the diseased plants in the greenhouse. Possibly insect injury favors attack by serving as an avenue of entrance for the fungus or by weakening the host plant.

Literature Cited

1. Campbell, W. A., and J. T. Presley. 1946. Diseases of cultivated guayule and their control. U. S. Depart. Agr. Circ. 749: 1-42.
2. Campbell, W. A., L. D. Leach, J. T. Presley, and W. C. Snyder. 1943. Some diseases of guayule in California. Plant Dis. Reptr. 27: 63-66.
3. Presley, J. T., and L. Weston. 1944. Fungi isolated from diseased guayule. Plant Dis. Reptr. 28: 984-985.

DEPARTMENT OF PLANT PHYSIOLOGY AND PATHOLOGY, TEXAS AGRICULTURAL EXPERIMENT STATION; AND FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, COLLEGE STATION, TEXAS.

RESISTANCE TO BLACK SHANK OF CERTAIN FLUE-CURED TOBACCO VARIETIES IN SEEDLING AND TRANSPLANT STAGES

G. B. Lucas and F. A. Todd

Abstract

Laboratory, greenhouse and plant bed tests of 7 varieties and strains of tobacco showed wide ranges in levels of resistance to black shank (caused by Phytophthora parasitica (Dast.) var. nicotianae (Breda de Haan) Tucker). Seedlings in the two- to four-leaf stage and plants of transplant size were inoculated with either zoospores or mycelium of the fungus. Strain T x L (Nicotiana tabacum x N. longiflora) was immune. Oxford 1, 400; and strain TL 106 (from the same source as T x L) were highly susceptible in both the seedling and transplant stage. Oxford 3, Dixie Bright 101, and Dixie Bright 102 were more resistant in the transplant stage than in the seedling stage. Plants of transplant size of both Dixie Bright 101 and Dixie Bright 102 were more resistant than Oxford 1 and Oxford 3; Dixie Bright 101 was as resistant as Dixie Bright 102. In plant bed tests on black-shank infested soil no diseased plants of Dixie Bright 101 or Dixie Bright 102 were seen, whereas, in some plots of the same tests all plants of the 400 variety were killed.

The black shank (Phytophthora parasitica (Dast.) var. nicotianae (Breda de Haan) Tucker) resistant Oxford varieties of flue-cured tobacco were released in North Carolina in 1943 (2). Observations on the performance of these varieties led to the assumption they were susceptible in the seedling stage and that the level of resistance increased with plant age. Although experimental data were lacking, this belief was perpetuated until it became widely accepted. Later, Apple (1), conducting field tests with certain susceptible and resistant varieties, showed the incidence of black shank was greater in plants inoculated with the causal organism at transplanting time than in those inoculated later in the growing season. Abundant data are available on field resistance of various black shank-resistant varieties of tobacco (4). However, there is little published information on their resistance in the seedling stage. The results of inoculation experiments with several varieties and strains of tobacco in both seedling and transplant stages are herein presented. A preliminary report has been published (3).

MATERIALS AND METHODS

The seven varieties or strains of tobacco used and their field resistance to black shank were as follows: 1) 400, susceptible; 2) Oxford 1, moderately resistant; 3) Oxford 3, highly resistant; 4) Dixie Bright 101, moderately resistant; 5) Dixie Bright 102, highly resistant; 6) TL 106[1], susceptible; 7) TxL[1], highly resistant. Seeds were sown in 4-inch pots of sterilized compost. When the seedlings were in the two- to four-leaf stage they were thinned to approximately 100 seedlings per pot. These were inoculated by pouring over them 20 ml. of a zoospore suspension of the black-shank fungus.

The zoospores used for inoculation were produced by floating in water in a Petri plate bits of aerial mycelium taken from two-week-old oatmeal agar cultures. The Petri plate was placed in a dark incubator at 30°C. Usually within two to three days sporangia were produced abundantly. If plates with sporangia were chilled in an 8°C. refrigerator for 30 minutes and then exposed to light at room temperature (21° C), the sporangia produced zoospores in great numbers within a few minutes. Following inoculation, the seedlings were placed in an incubator at about 30° and illuminated continuously with daylight fluorescent light. After three to four days counts were made of the number of seedlings killed in each pot. This test was repeated five times.

Another test was made as follows: Individual seedlings with leaves about 1 in. in diameter were transplanted to thumb pots and grown for some time in the greenhouse. When they reached

---

[1] T L 106 is a wildfire-immune selection from Nicotiana tabacum x N. longiflora and backcrossed to N. tabacum. T x L is tetraploid N. tabacum x N. longiflora. The seed were obtained from Dr. E. E. Clayton.

the approximate size of a tobacco transplant (about two to three weeks) they were inoculated with the black-shank fungus. Usually four plants of each variety were inoculated. Either mycelium or zoospores was used as inoculum and both proved equally effective. If mycelium was used, the fungus was grown on oatmeal agar in Petri plates until abundant aerial mycelium was produced. Then a 1/4 x 1/2 in. piece of the culture was cut out and inserted into a hole 1 inch deep in the soil at the base of the transplant. If zoospores were used as inoculum, about 10 ml. of a zoospore suspension were placed in the hole in the soil at the base of each plant. The inoculum was covered and the plants were placed in the 30° C incubator. After three to four days the plants were examined for black shank symptoms. This test was repeated eight times with similar results.

Additional information was obtained in connection with plant bed tests with various chemical soil treatments for black shank control in the seed bed. The plots (10 sq. yds. each) were divided in half and seeded to two varieties of tobacco. A black-shank susceptible variety, 400, was seeded on one-half the plot, and a black-shank resistant variety on the other. Over a three-year period, Dixie Bright 101 was used in eight tests in five different counties and Dixie Bright 102 was used in two experiments in one county. Stand and disease counts were made and the varieties observed for black shank symptoms.

RESULTS

The comparative disease resistance of the 7 varieties as very young seedlings (two- to four-leaf stage) and in the transplant stage of growth is shown in Figure 1.

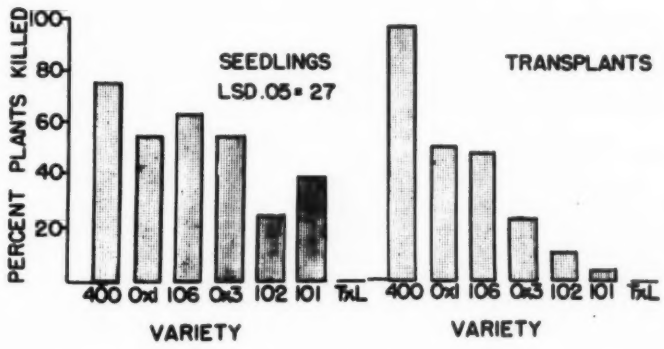


FIGURE 1. Left, comparative black-shank resistance of 7 varieties and strains of tobacco in the 2- to 4-leaf stage; average of 5 tests. Right, comparative black-shank resistance in the transplant stage; average of 8 tests.

The T x L strain was highly resistant. No diseased plants were seen in any of the tests. Oxford 1, variety 400, and TL 106 showed little resistance in either the seedling or transplant stage. Oxford 3, Dixie Bright 101, and Dixie Bright 102 were more resistant in the transplant stage than in the seedling stage. Dixie Bright 102 was more resistant in the seedling stage than any other variety. In plants of transplant size, both Dixie Bright 101 and Dixie Bright 102 were more resistant than Oxford 1 and Oxford 3.

In the plant bed experiments, no diseased Dixie Bright 101 or Dixie Bright 102 plants were seen in any of the 10 tests over a three-year period. In some of these plots, however, all plants of the susceptible variety, 400, were killed.

DISCUSSION

The results of these greenhouse, laboratory, and plant bed tests are interesting from several aspects. The tests show that different levels of resistance to black-shank can be demon-

strated in tobacco varieties in both seedling and transplant stages. Not all varieties are highly susceptible in the plant bed. The T x L strain is highly resistant to black shank and the very young seedlings show no symptoms of the disease. Variety 400, in contrast, is susceptible to black shank from seedling to mature plant. Neither Oxford 1 nor Oxford 3 is as resistant in the seedling and transplant stages as Dixie Bright 101 or Dixie Bright 102. Also, it appears that the resistance of Dixie Bright 101 in the transplant stage is equal to that of Dixie Bright 102, even though the latter has higher field resistance than the former variety.

It is surprising that Dixie Bright 101 and 102 are more resistant in the seedling and transplant stages than Oxford 1 and 3, since the field resistance of Dixie Bright 101 is equal to that of Oxford 1 and Dixie Bright 102 is comparable to Oxford 3. It has been shown there is linkage between the factors controlling bacterial wilt resistance and those controlling black shank resistance (5). It may be that the increased resistance in the seedling and transplant stage of Dixie Bright 101 and Dixie Bright 102 is due to the presence of accessory genes derived from TI 448 A, the wilt-resistant parent of these varieties. Even though TI 448 A has little resistance to black shank these genes may contribute to the over-all black-shank resistance of Dixie Bright 101 and Dixie Bright 102.

SUMMARY

Seven varieties and strains of tobacco were tested for black shank resistance in the seedling and transplant stage. Strain T x L was highly resistant. Oxford 1, variety 400, and strain TL 106 were highly susceptible in both the seedling and transplant stage. Oxford 3, Dixie Bright 101, and Dixie Bright 102 were more resistant in the transplant stage than in the seedling stage. When the plants were of transplant size both Dixie Bright 101 and Dixie Bright 102 were more resistant than Oxford 1 and Oxford 3; Dixie Bright 101 was as resistant as Dixie Bright 102.

In plant bed tests, over a three-year period, no black shank infected plants of Dixie Bright 101 and Dixie Bright 102 were seen, whereas, in some plots of the same tests all plants of the black-shank susceptible variety, 400, were killed.

Literature Cited

1. Apple, J. L. 1953. The evaluation of certain field inoculation techniques with Phytophthora parasitica var. nicotianae on resistant and susceptible varieties of tobacco. Master of Science Thesis. North Carolina State College.
2. Bullock, J. F., and E. G. Moss. 1943. Strains of flue-cured tobacco resistant to black shank. United States Dept. Agr. Circ. 682.
3. Lucas, G. B., and E. L. Moore. 1952. Black shank control in tobacco plant beds. (Abstr.) Phytopath. 42: 285-286.
4. Moore, E. L., G. B. Lucas, and E. E. Clayton. 1952. Granville wilt and black shank resistant tobacco varieties. North Carolina Agr. Exp. Sta. Bul. 378.
5. Smith, T. E., and E. E. Clayton. 1948. Resistance to bacterial wilt and black shank in flue-cured tobacco. Phytopath. 38: 227-229.

PLANT PATHOLOGY SECTION, NORTH CAROLINA STATE COLLEGE AND UNITED STATES DEPARTMENT OF AGRICULTURE, SECTION OF TOBACCO, MEDICINAL AND SPECIAL CROPS.

A NORTH AMERICAN RACE OF CROWN RUST ATTACKING THE OAT VARIETIES LANDHAFER AND SANTA FE[1]

M. D. Simons

Crown rust (Puccinia coronata) is the most serious disease of oats in the major oat producing regions of the United States. The most effective control measure for this disease is the breeding of resistant varieties. Good sources of resistance to the North American races of the crown rust fungus are relatively few in number, and in recent years, many experiment stations have been relying heavily on two resistant varieties, Landhafer and Santa Fe. Until recently these two varieties have been resistant, in the seedling stage, to all North American collections of crown rust to which they have been tested.

Early in 1954, in the course of identifying races of crown rust in connection with the annual survey, two collections appeared to differ markedly from any race that had been previously seen in North America. Both Landhafer and Santa Fe were fully susceptible in the seedling stage. The varieties Trispernia and Bondvic, which had always before shown at least a certain amount of resistance, also were completely susceptible. Anthony, Appler, Bond, and Ukraine were susceptible. Victoria and Saia were the only varieties among the new set of differentials that were resistant.

The two unusual collections of crown rust, originating in the nursery at Winnipeg, Manitoba, had been received the previous summer from Drs. B. Peturson and W. F. Hanna. In view of the rarity of this race, there was a possibility that it would not overwinter. Consequently, it appeared advisable to cease carrying it on living plants in the greenhouse before there could be any chance of its escaping into the field onto spring-sown oats. For this reason, the available urediospores were used, without purification, to run a re-check on the differential varieties. The reactions were similar to those mentioned above, and the race was described on that basis as a new race.[2]

A number of tests carried out a short time later, however, indicated that the new race did not attack Ukraine. The original collections apparently had consisted of a mixture of the new race, to which Ukraine is rather highly resistant, with a race attacking Ukraine. On this basis, the new race must be described as number 263, which when tested on the differential varieties, corresponds to a race identified by Vallega[3] in Argentina.

In the limited time available, a number of older varieties and strains, and certain varieties currently of interest commercially or in breeding programs, were tested in the seedling stage for reaction to the new race in an attempt to find readily available sources of resistance. The older varieties included many that have been used or considered as sources of resistance to crown rust in the past. In addition to these, the entries of the 1954 Uniform Rust Nursery, the 1954 Uniform North Central and Spring Sown Red Oat Yield Nurseries, and certain newly discovered sources of resistance were tested.

The results were disappointing. With the exception of Victoria and Ukraine, all entries of the Uniform Rust Nursery were susceptible. All entries of the North Central Uniform Yield Nursery were susceptible with the possible exception of Vicland x (Branch x (Clinton[2] x Santa Fe)) (C. I. 6916). Osage (C.I. 3991) was the only resistant entry found in the Uniform Spring Sown Red Oat Yield Nursery.

Nearly all of the resistant varieties or strains (Table 1) of cultivated species could either be traced directly to Ukraine or Victoria or else exhibited reactions strongly resembling the characterisitic "hypersensitive" Victoria type reaction associated with susceptibility to Helminthosporium victoriae. The varieties Saia and Glabrota were highly resistant to both the new race and to H. victoriae, but belong to the 7-chromosome species, Avena strigosa.

Summary

Two isolates of race 263 of crown rust of oats have been identified from material obtained from Manitoba, Canada. This race vigorously parasitized the varieties Landhafer, Santa Fe,

[1]Cooperative investigation between the Field Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture, and the Iowa Agricultural Experiment Station. Journal paper No. J-2540 of the Iowa Agricultural Experiment Station, Ames, Iowa. Project No. 1176.

[2]Simons, M. D. 1953. National Oat Newsletter (Sponsored by the National Oat Conference), 4:37-38, 1954.

[3]Unpublished data furnished by J. Vallega.

Table 1. Reaction of the new standard differential varieties and of certain other varieties and strains of oats to a North American isolate of race 263 of crown rust.

Variety	C.I. or P. I. No.	Reaction type or types[a]
Anthony	CI 2143	4
Victoria	CI 2401	2
Appler	CI 1815	4
Bond	CI 2733	4
Landhafer	CI 3522	4-
Santa Fe	CI 4519	4
Ukraine	CI 3259	0
Trispernia	CI 4009	4
Bondvic	CI 5401	4
Saia (Avena strigosa)	CI 4639	I, 0
Avena Victoria (Scasso)	CI 2764	2
Avena 64s (Pampa)	CI 2767	0, 1, 2
Common of the Country (Red Algerian)	CI 2861	0, 1, 2
64q	CI 2927	2
64r	CI 2928	2
64t	CI 2930	1, 2
Klein Mar	CI 3155	1
Arkansas 674	CI 4529	I, 0
Garry (old)	CI 4801	2, 3-
Tama	CI 3502	2
Alamo	CI 5371	2
Glabrota (Avena strigosa)	CI 2630	I
Ascencao	PI 186603	2
Ascencao	PI 189624	2
Hybrido F_2 au 12-M-3-R. 47	PI 174513	2

[a]"I" indicates no macroscopic evidence of infection; "O", necrosis or chlorosis, but no uredia; "1", necrotic or chlorotic areas, a few of which contain small uredia; "2", necrotic or chlorotic areas, most of which contain small to mid-sized uredia; "3", chlorotic areas surrounding abundant, mid-sized uredia, no necrosis; "4", no necrosis or chlorosis, uredia large and abundant.

Trispernia, and Bondvic. Ukraine and Victoria were resistant and offer possible sources of resistance. The Avena strigosa varieties, Saia and Glabrota, were highly resistant.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE AND THE IOWA AGRICULTURAL EXPERIMENT STATION, AMES, IOWA.

DISEASES OF PEARL MILLET IN GEORGIA[1]

E. S. Luttrell[2]

SUMMARY

The most common foliage diseases of pearl millet (Pennisetum glaucum (L.) R. Br.) in Georgia are Helminthosporium leaf spot (Helminthosporium stenospilum Drechs., H. sacchari (van Breda de Haan) Butler) and Cercospora leaf spot (Cercospora penniseti Chupp). These, and other diseases, usually appear late in the season and have little effect on forage production. Seed production is often seriously affected by head mold. The principal head mold fungi are Oidium tenellum (Berk. & Curt.) Linder, Helminthosporium sacchari (or H. stenospilum), H. rostratum Drechs., Curvularia lunata (Wakker) Boed., and Fusarium moniliforme Sheld. Minor diseases are zonate leaf spot (Gloeocercospora sorghi Bain & Edgerton), zonate eye spot (H. rostratum), Rhizoctonia blight (Rhizoctonia solani Kuehn, R. zeae Vorhees), southern blight (Sclerotium rolfsii Sacc.), rust (Puccinia penniseti Zimm.), and leaf mold (Curvularia lunata, C. geniculata (Tracy & Earle) Boed., C. maculans (Bancroft) Boed.). The symptomatology and etiology of these diseases are discussed. Diseases reported in other areas but not yet found in Georgia are listed.

Pearl millet (Pennisetum glaucum (L.) R. Br.) is one of the most important grasses for temporary summer grazing in Georgia, particularly in the Coastal Plain where it is most extensively grown (28). Although it is subject to a number of diseases (15), these diseases usually appear late in the season after the plants begin to head. As Burton (5) has pointed out, the young growth is remarkably free of diseases. Diseases, therefore, have little effect on forage production except on second growth late in the season. After the plants head, they generally become heavily infected with Helminthosporium and Cercospora leaf spots. Zonate leaf spot is potentially serious but rarely occurs in more than trace amounts. Minor diseases are Rhizoctonia blight, southern blight, zonate eye spot, leaf mold, and rust. Although the leaf spots probably affect seed production to some extent, the most important disease in this respect is head mold, which is usually prevalent by seed harvest time.

Since diseases have little effect on forage production, resistant varieties are not essential, although resistance to Helminthosporium leaf spot and Cercospora leaf spot might be desirable. The new variety "Starr", introduced by Burton and DeVane (6), appears to be no more resistant to diseases than common pearl millet. It has become popular because of its improved agronomic characters. Control of head mold is desirable, if seed is to be grown in this area. The heads are sometimes harvested by hand and cured in the barn, but this is a laborious process. It may prove necessary to resort to spraying or mechanical harvesting and artificial drying or to grow seed in areas in which head mold does not occur.

Few observations have been made on seedling diseases. The relationship of seed-borne fungi to poor germination, poor emergence, and seedling diseases should be investigated. Since millet is an annual crop, seed treatment also might be profitably investigated.

Helminthosporium Leaf Spot (Helminthosporium stenospilum Drechs. and H. sacchari (van Breda de Haan) Butler). -- Helminthosporium leaf spot is the most common and most damaging of the leaf diseases of millet. Although some infection has been observed as early as July, the disease usually develops extensively only in August, September, and October, after the plants head. The leaves may be severely blighted by the time the plants are mature. The causal fungus also is commonly associated with head mold. Helminthosporium leaf spot occurs more abundantly in the Coastal Plain than in the Piedmont.

The symptoms (Fig. 1) are extremely variable. They differ to some extent on different strains of millet, but various types of lesions may occur on a single leaf. The lesions vary from brown flecks, fine linear streaks, and small oval spots to large, irregular, oval, oblong, or almost rectangular spots measuring 1-10 x 0.5-3 mm. Sometimes large fusiform lesions

---

[1]Journal Series Paper No. 263, Georgia Experiment Station, Experiment, Georgia.
[2]With the collaboration of Dr. Glenn W. Burton, Earl H. DeVane, and Dr. Homer G. Wells.

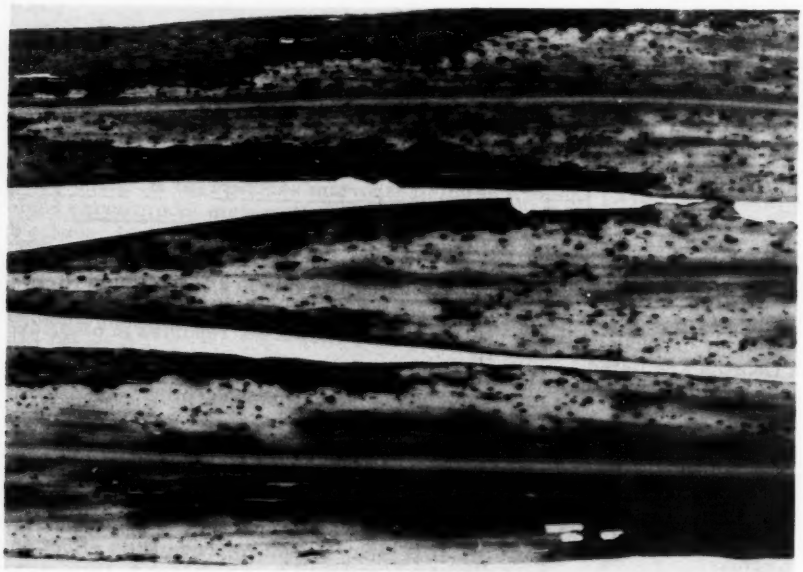


FIGURE 2. Cercospora leaf spot of pearl millet (Cercospora penniseti).

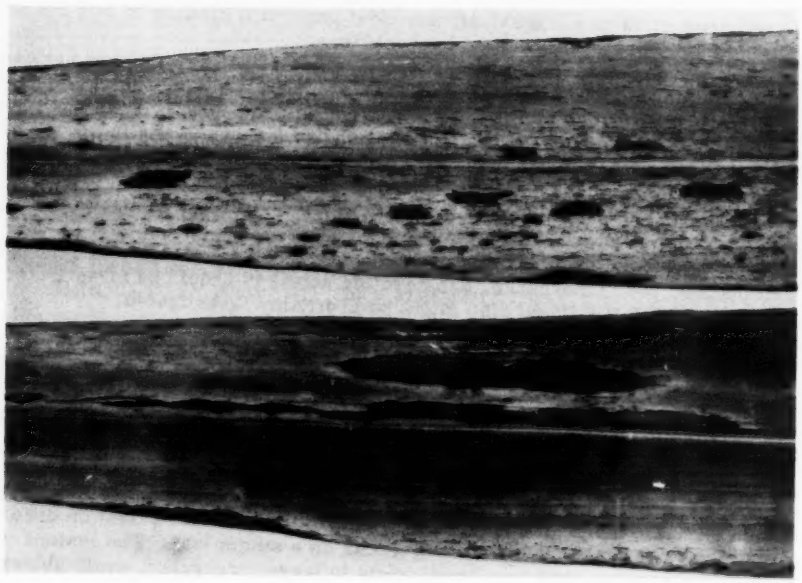


FIGURE 1. Helminthosporium leaf spot of pearl millet (Helminthosporium stenospilum or H. saccharri).

resembling those caused by H. turcicum on corn are produced. The spots may expand and coalesce to form long streaks 20-90 x 2.5-5 mm. The lesions may be solid dark brown but usually become tan or grayish brown except for a more or less distinct dark brown border. The surfaces, especially in moist weather, may be covered with a dense grayish-brown felt of conidiophores and conidia. The causal fungus also forms black wooly mats over individual grains in the heads (Fig. 5, left).

The conidia are fusoid to obclavate-fusoid, straight to usually slightly curved, thin-walled but often becoming moderately thick-walled at maturity, sub-hyaline, fuligenous, to pale or finally moderately dark fuligenous or olivaceous brown, 44-151 x 10.6-19.6 μ (means 66.8-108 x 12.3-16.6 μ), and 4-13-septate (means 7.6-9). The largest conidia have been found on mats on the heads. In one collection the conidia were unusually thick-walled (1.5-2.9 μ thick), darker olivaceous brown, 39-90 x 12.3-16.8 μ (mean 70 x 15.4 μ), and 4-10-septate (mean 8.3).

Isolates of the fungus produced abundant infection on pearl millet (Starr) in the greenhouse when plants that had headed were inoculated. It was difficult or impossible to obtain infection of younger plants. This should be taken into account in any experimental work on this or other diseases of pearl millet. When inoculated on sugar cane (Lahi Pink Striped), the isolates from millet produced typical brown stripe lesions. Isolates from brown stripe lesions on sugar cane produced leaf spot on millet. No isolate from millet produced eye spot lesions on sugar cane, and a single isolate from eye spot of sugar cane failed to infect millet. The millet Helminthosporium seems to be morphologically and pathologically similar to the fungus causing brown stripe of sugar cane, which has generally been referred to H. stenospilum. However, Parris (23) has stated that both H. stenospilum and H. sacchari are associated with brown stripe of sugar cane. He distinguished H. stenospilum from H. sacchari by its much thicker conidial walls. If Parris's separation of these two species is accepted, only the single collection on millet with thick conidial walls could be referred to H. stenospilum. All others would be placed in H. sacchari. It should be noted that both Parris's isolate and the collection on millet, which according to Parris represent H. stenospilum, agree with Drechsler's (11) original description of this species only in the thick-walled conidia. The conidial dimensions are much smaller than those given by Drechsler, who mentioned the larger size of the conidia as one of the characters distinguishing H. stenospilum from H. sacchari. Also, Parris's conclusion that H. sacchari causes both eye spot and brown stripe was not supported by inoculations. It is evident that some confusion exists in the taxonomy of the Helminthosporia on sugar cane. Until this situation is clarified, it is difficult to determine the proper name to apply to the millet Helminthosporium.

Zonate Eye Spot (Helminthosporium rostratum Drechs.) -- This disease was reported on pearl millet in Georgia by Young, Lefebvre, and Johnson (30). Zonate eye spot has not been found in this survey, although the causal fungus, H. rostratum, has been found associated with head mold.

Cercospora Leaf Spot (Cercospora penniseti Chupp). -- Cercospora leaf spot occurs regularly in September and October, developing even later in the season than Helminthosporium leaf spot. In the Coastal Plain it appears to be a minor disease, although in this area it may simply be obscured by the heavy development of Helminthosporium leaf spot. It is usually the most serious foliage disease in the Piedmont.

The spots on the leaves (Fig. 2) are occasionally oblong to rectangular but typically are oval and 1-8 x 0.8-2.5 mm. The margins are dark brown, the centers pale tan to gray or chalky white and dotted with rows of black conidiophore tufts. In moist weather the spots are covered with silvery layers of conidia. Spots occasionally occur on the stems. These spots measure 1-5 x 0.5-2 mm. and are similar to those on the leaves. Sometimes dark brown runners extend longitudinally from the ends of these spots for as much as 3 mm. (Fig. 3).

The conidiophores arise in clusters of more than a dozen from small, brown, substomatal stromata. They are brownish-fuligenous, paler toward the tips which bear circular conidial scars and are geniculate, 2-12-septate, and measure 30-434 x 2.8-5.3 μ at the tips, 3.1-5.3 μ in the middle, and 5.0-6.4 μ at the base. Under moist conditions they become greatly elongated and nearly straight. The conidia are acicular to almost filiform, widest in the basal cell or in the first 2 to 3 cells, hyaline, multiseptate, and 36-305 x 2.8-5.3 μ (means 91-136 x 4.0-4.3 μ).

The few inoculations attempted with macerated mycelium from cultures of the fungus were unsuccessful.

The millet fungus has been named Cercospora penniseti by Chupp (9). However, it is very closely related morphologically to C. apii Fres. In view of the range of pathogenicity demonstrated for C. apii by Johnson and Valleau (16), C. penniseti should be carefully compared with

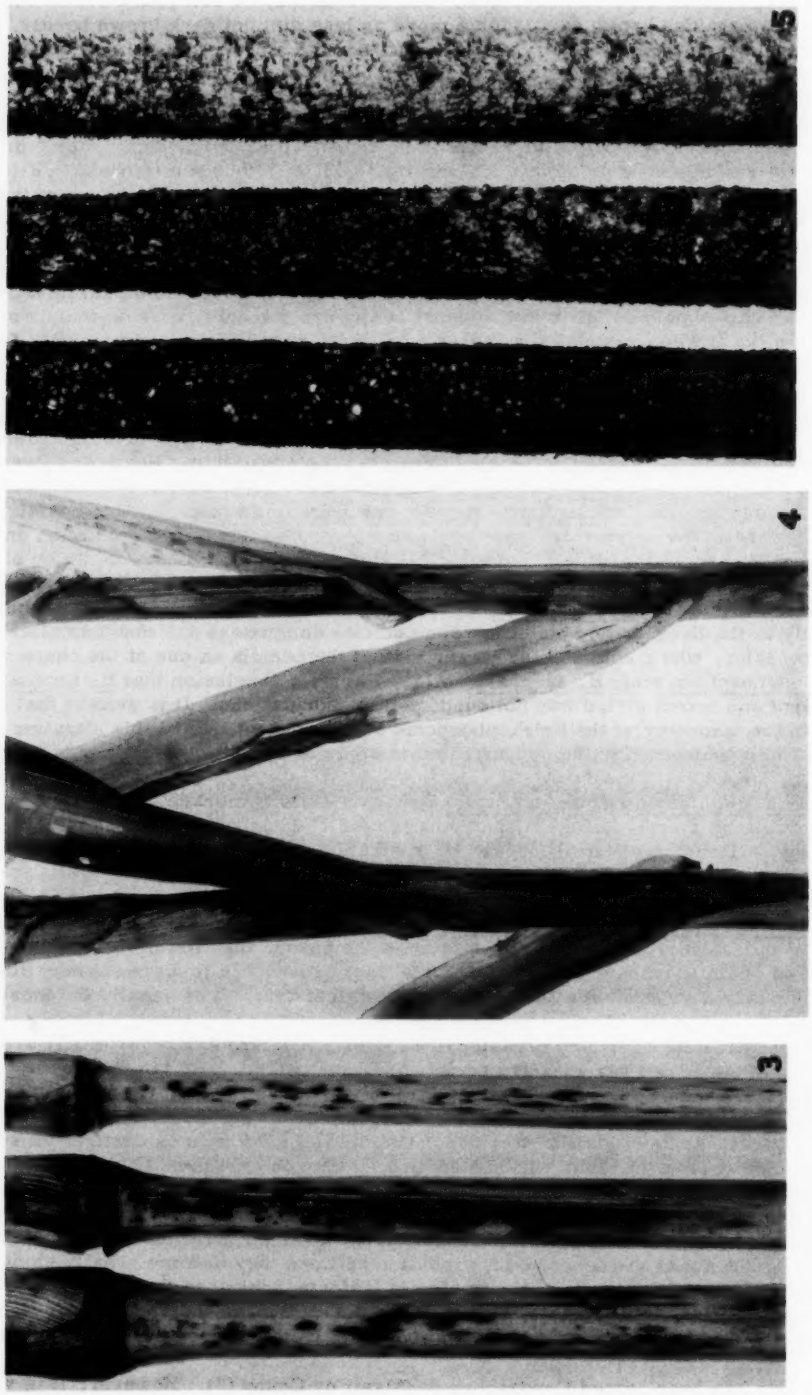


FIGURE 3. Lesions on stems of pearl millet produced by Cercospora penniseti.
FIGURE 4. Rhizoctonia blight of pearl millet (Rhizoctonia solani).
FIGURE 5. Head mold of pearl millet. Left. Black mold caused by Helminthosporium and Curvularia spp. Middle. Orange mold caused by Fusarium sp., mixed with black mold. Right. White mold caused by Oidium tenellum.

C. apii. These species probably are synonymous.

Zonate Leaf Spot (Gloeocercospora sorghi Bain & Edgerton). -- Although zonate leaf spot may cause appreciable damage on millet, it usually is of minor importance. This disease was conspicuous in a single planting of millet at Tifton in August 1949. Only traces of it have been found at Tifton and in other localities in subsequent years.

The lesions appear as water-soaked spots which develop tan centers and dark brown borders and measure 3.5-5 x 2-3.5 mm. The spots enlarge to form roughly orbicular or semi-circular blotches covering half or more of the width of the leaf. The blotches are various shades of dark brown mottled with pale tan spots. The tan areas tend to be arranged in circles and give the appearance of incomplete tan rings alternating with dark brown rings. Often, especially on narrow-leaved strains of millet, this zonation fails to develop. In moist weather tiny salmon-colored globules are visible under the hand lens on both surfaces of the spots. As the leaves die, lenticular black sclerotia appear in the dead tissue. The sclerotia are less abundant than on sorghum.

The conidia are nearly identical with those described on sorghum (1, 19). They arise on short conidiophores from small, plectenchymatous, erumpent stromata and are attenuate-obclavate, hyaline, multiseptate, and 25-104 x 2.0-2.5 μ (mean 74.5 x 2.2 μ).

Inoculations have been inconclusive. An isolate of Gloeocercospora sorghi from sorghum caused severe infection of sorghum but only slight infection of millet. An isolate from millet caused only moderate infection of either sorghum or millet. Further inoculations should be made to determine whether the strain on millet is identical with the one on sorghum.

Rhizoctonia Blight (Rhizoctonia solani Kuehn and R. zeae Voorhees). -- Rhizoctonia blight occasionally occurs on millet, and sometimes traces of this disease may be found throughout an entire planting. It apparently causes little damage, although occasionally individual tillers may be killed, and rarely an entire plant may be blighted so severely that it has the appearance of being scalded. The disease occurs more frequently on Starr millet. It is doubtful that common pearl millet is more resistant. It probably escapes infection because of its tall loose growth, whereas the low dense habit of Starr millet creates conditions more favorable for the growth of the fungus.

The lesions (Fig. 4) occur chiefly on the leaf sheaths and base of the blade. They are irregular blotches 4-7 cm. long which are at first water-soaked but bleach to pale tan with an indistinct brown border. A diffuse white to tan mycelial mat covers the surface of the lesions and adjacent green tissue. Old blotches may be peppered with tiny brown sclerotia.

Rhizoctonia solani is usually associated with these lesions. Isolates of this fungus were of two types. One formed a sparse tan mycelium and large globular dark-brown sclerotia. The other produced no discrete sclerotia but formed a tan stromal-like layer over the surface of the agar. Isolates of both types proved highly virulent, causing severe aerial infection and damping off when inoculated on a series of grasses and legumes.

Rhizoctonia zeae was found once at Tifton in 1951. It occurred on the same type of lesions and was mixed with R. solani. It was distinguished in the field by the presence of tiny, globular, red sclerotia on the surface of the lesions. In culture R. zeae formed a white mycelium and spherical red sclerotia embedded in the surface and immersed throughout the agar. Isolates caused mild infection on several grasses but failed to attack legumes.

Southern Blight (Sclerotium rolfsii Sacc.). -- Southern blight may kill single tillers and occasionally entire plants but rarely occurs on millet. The white mycelial mats of the fungus spread upward inside of the leaf sheaths as much as a foot above the ground. The tissue beneath the mat becomes discolored and is eventually killed and shredded. Sclerotia form inside of the leaf sheaths and around the base of the plant as on sorghum (19).

Rust (Puccinia penniseti Zimm.). -- Rust is so rare and inconspicuous that it has been found only by accident when examining leaves with a hand lens. It was found on a few leaves at Tifton in October 1949 and again in 1953. There are no conspicuous lesions. The only evidence of infection is tiny rust pustules scattered over both surfaces, but more abundantly on the upper surface of the leaves.

Only the uredial stage of the fungus has been observed. The uredia appear as tiny pale orange-brown pustules which rupture the epidermis. The uredospores are borne on hyaline pedicels measuring 36-61 x 7.6-10.6 μ. They are ovoid, or less commonly oblong or obpyriform, 1-celled, medium yellowish-brown, echinulate, and 34-50 x 26-31 μ (mean 37.3 x 29.3 μ).

and Curvularia spp. Middle. Orange mold caused by Fusarium sp., mixed with black mold. Right. White mold caused by Oidium tenellum.

The uredial stage corresponds closely with the revised description of Puccinia penniseti given by Ramakrishnan and Soumini (25). Until more adequate material is available the millet fungus is referred to this species. Although P. penniseti has not previously been reported in the United States, P. cenchri Diet. & Holw., which has been reported on Pennisetum multiflorum (27), seems to be a closely related species.

Leaf Mold (Curvularia spp.). -- Pearl millet is a rich source of Curvularia spp. They appear abundantly on leaves placed in a moist chamber in association with other leaf spotting fungi and sometimes alone on various types of spots and blotches. No inoculations have been made, and they are assumed to be secondary organisms. However, their pathogenicity should be tested since Mitra (21) reported slight infection of millet with Curvularia lunata (Wakker) Boed. from sorghum and demonstrated the pathogenicity of a related fungus from pearl millet which he named Acrothecium penniseti Mitra (Curvularia penniseti (Mitra) Boed.). Representatives of all three of Boedijn's (4) sections of the genus have been found on millet.

Curvularia lunata is the most common species. The conidia are broad clavate, 3-septate, with the third cell from the base greatly swollen, the two middle cells dark fuligenous, the terminal cells pale, inequilateral to sharply curved at the swollen third cell, 22-30 x 10.6-14.3 μ (mean 27.0 x 12.9 μ), length/width ratio 2.1. C. penniseti, which is reported (21) to cause oval or oblong brown spots with yellow margins and also to infect the heads of millet in India, is distinguished from C. lunata by its larger conidia. Groves and Skolko (13) recorded the conidial dimensions of C. penniseti as 30.4-41.8 x 17.1-20 μ, as given by Mitra (21) in his diagnosis of this species. However, in the text of his paper Mitra reported the dimensions as 25-41.8 x 12.5-20 μ. Mitra also stated that the conidia were highly variable in culture on different media. The smallest conidia were produced on potato juice agar. These measured 15-20 x 10-12.5 μ and were smaller than those usually produced by C. lunata. If such variation occurs in the field, it will be difficult to separate these two species.

Curvularia geniculata (Tracy & Earle) Boed. occurs less frequently than C. lunata. The conidia are fusoid, 3-6-(usually 4)-septate, swollen in the middle (usually in the third or fourth cell from the base), the middle cells dark fuligenous or olivaceous fuligenous, the smaller terminal cells paler, often subhyaline, inequilateral to strongly curved, 27-39 x 8.7-11.8 μ (mean 32.5 x 10.6 μ), length/width ratio 3.1. These conidial dimensions correspond closely to those given for C. affinis Boed. (13). Nevertheless, the millet fungus is referred to the older species C. geniculata since it seems doubtful that C. affinis can be maintained as a distinct species on the basis of conidial size.

Curvularia maculans (Bancroft) Boed. has been found only once on millet. The conidia are straight, broad-elliptical, 3-septate, the middle septum thickened and dark, both intercalary cells swollen and dark fuligenous to olivaceous, the smaller terminal cells pale, 19-25 x 9.2-11.8 μ (mean 21.6 x 10.6 μ), length/width ratio 2.0.

Head Mold. -- Head mold, with which various fungi are associated, occurs in nearly every planting grown for seed and develops extensively if the weather is moist (Fig. 5). These fungi destroy an appreciable portion of the grain and probably are largely responsible for the poor germination of the seed harvested. The damage is increased by the attacks of insects. The fungi cover individual grains or entire heads. They usually occur in mixtures, although often a single species predominates on individual heads or throughout an entire planting.

One of the commonest and most conspicuous, although probably the least damaging, fungi is Oidium tenellum (Berk. & Curt.) Linder (18). This fungus apparently grows primarily on the glumes and on grains damaged by insects. It forms a mealy white coating over the heads (Fig. 5, right). Under the hand lens, the heads appear covered with white hairs bearing white beads at the tips. These are erect, hyaline conidiophores thickly covered with short, spine-like sterigmata in club-shaped areas at the tips. The conidia are ovoid, hyaline, non-septate, and 12-23 x 8.4-13.7 μ (mean 17.8 x 11.0 μ). This same species commonly occurs on heads of grain sorghum. It was erroneously reported in a previous paper (19) as "Corethropsis sp.".

A second type of mold covers individual grains with dense, black, wooly mycelial mats (Fig. 5, left). The fungi are Helminthosporium stenospilum (or H. sacchari), H. rostratum, and Curvularia lunata, all of which also occur on the leaves.

In a third type the individual grains are caked with orange crusts (Fig. 5, middle). This type is produced by a Fusarium sp., probably F. moniliforme Sheld.

Diseases Not Yet Reported in Georgia. -- The most important diseases of pearl millet in Georgia, Helminthosporium leaf spot and Cercospora leaf spot, apparently do not occur, or have received little attention, in India and Africa where this plant has long been one of the chief grain crops. On the other hand, the most important diseases in these countries have not yet been reported in Georgia.

1. Smut is probably the most important disease in India and Africa. The principal species is Tolyposporium penicillariae Bref. (3). This was considered a synonym of Ustilago penniseti (Kunze) Rabh. by Ciferri (10). A second species is T. senegalense Speg., which is synonymous with T. bullatum (Schroet.) Schroet. (14). Also doubtfully reported are Neovossia barclayana Bref. (8) and Tilletia ajrekari Mundkur (22).
2. Downy mildew caused by Sclerospora graminicola (Sacc.) Schroet. (29) is the second most important disease in India and Africa.
3. Piricularia leaf spot caused by Piricularia sp. in India (20).
4. Bacterial leaf spot caused by Pseudomonas syringae v. Hall (syn. Bacterium holci Kendrick) in Iowa (17).
5. Fusarium inflorescence blight and seedling blight caused by Fusarium roseum Lk. (?) in Africa (12).
6. Top rot ("pokka bong") caused by Fusarium moniliforme in India (24).
7. "Asali disease" or "sugary disease" caused by Sphacelia sp. in Africa (26).
8. Inflorescence blight caused by Balansia claviceps Speg. in Africa (8).
9. "Freckled yellow" and "stripe" diseases caused by viruses in India (7).
10. Chlorosis resulting from zinc deficiency in Florida (2).

Literature Cited

1. Bain, D. C., and C. W. Edgerton. 1943. The zonate leaf spot, a new disease of sorghum. Phytopath. 33: 220-226.
2. Barnette, R. M., et al. 1936. The use of zinc sulphate under corn and other field crops. Bull. Florida Agric. Exp. Sta. 292: 1-51.
3. Bhatt, R. S. 1946. Studies in the Ustilaginales. I. The mode of infection of the Bajra plant (Pennisetum typhoides Stapf & Hubbard) by the smut Tolyposporium penicillariae Bref. Jour. Indian Bot. Soc. 25: 163-186. (R.A.M. 26: 200-201. 1947).
4. Boedijn, K. B. 1933. Ueber einige phragmosporen Dematiazeen. Buitenzorg Jard. Bot. Bull. Ser. III, 13: 120-134.
5. Burton, G. W. 1951. The adaptability and breeding of suitable grasses for the southeastern states. In Advances in Agronomy Vol. 3, Academic Press, New York, pp. 197-241.
6. Burton, G. W., and E. H. DeVane. 1951. Starr millet. Southern Seedsman 14 (3): 17, 68, 69.
7. Cherian, M. C., and M. S. Kylasan. 1937. Preliminary studies on the 'freckled yellow' and 'stripe' diseases of Cholam. Proc. Assoc. Econ. Biol., Coimbatore 1936, 4: 57-63. (R.A.M. 17: 169. 1938).
8. Chevalier, A. 1931. Une maladie du Pénicillaire au Sénégal. Rev. de Bot. Appliquée 11: 49-50. (R.A.M. 10: 450. 1931).
9. Chupp, C. 1954. A monograph of the fungus genus Cercospora. Ithaca, New York.
10. Ciferri, R. 1931. Quinta contribuzione allo studio degli Ustilaginales (No. 127-257). Ann. Mycol. 29: 1-74. (p. 57).
11. Drechsler, C. 1928. A species of Helminthosporium distinct from Helminthosporium sacchari, causing brown stripe of sugar cane (abst). Phytopath. 18: 135-136.
12. Fourneau, L. 1928. Note sur une affection cryptogamique du petit mil (Panicum spicatum Roxb.) causée par un hyphomycète. Rev. de Bot. Appliquée 8: 681-683. (R.A.M. 8: 234. 1929).
13. Groves, J. W., and A. J. Skolko. 1945. Notes on seed-borne fungi. III. Curvularia. Canadian Jour. Res. Sect. C, 23: 94-104.
14. Hirschhorn, E. 1941. Nota sinononimica: Tolyposporium senegalense es sinonimo de T. bullatum. Rev. Argent. Agron. 8: 384-386. (R.A.M. 24: 62. 1945).
15. Johnson, H. W. 1953. Leaf diseases of grasses in the South. In Plant Diseases, U. S. Dept. Agr. Yearbook of Agric. 1953. Washington, D. C. (p. 259-262).
16. Johnson, E. M., and W. D. Valleau. 1949. Synonomy in some common species of Cercospora. Phytopath. 39: 763-770.

17. Kendrick, J. B. 1926. Holcus bacterial spot of Zea mays and Holcus species. Iowa Agric. Exp. Sta. Res. Bull. 100.
18. Linder, D. H. 1942. A contribution towards a monograph of the genus Oidium (Fungi Imperfecti). Lloydia 5: 165-207.
19. Luttrell, E. S. 1950. Grain sorghum diseases in Georgia -- 1949. Plant Dis. Reptr. 34: 45-52.
20. Mehta, P. R., B. Singh, and S. C. Mathur. 1952. A new leaf spot disease of Bajra (Pennisetum typhoides Stapf and Hubbard) caused by a species of Piricularia. Indian Phytopath. 5: 140-143.
21. Mitra, M. 1921. Morphology and parasitism of Acrothecium Penniseti n. sp. (a new disease of Pennisetum typhoideum). Mem. Dept. Agric. India Bot. Ser. 11: 57-74. (R.A.M. 1: 103).
22. Mundkur, B. B. 1939. A contribution towards a knowledge of Indian Ustilaginales. Trans. British Mycol. Soc. 23: 86-121.
23. Parris, G. K. 1950. The Helminthosporia that attack sugar cane. Phytopath. 40: 90-103.
24. Ramakrishnan, T. S. 1941. Top rot ('twisted top' or 'pokkah bong') of sugar cane, sorghum, and Cumbu. Curr. Sci. 10: 406-408. (R.A.M. 21: 161. 1942).
25. Ramakrishnan, R. S., and C. K. Soumini. 1948. Studies in cereal rusts. I. Puccinia penniseti Zimm. and its alternate host. Indian Phytopath. 1: 97-103.
26. Ritchie, A. H. 1926. Entomological report, 1925-26. Rept. Dept. Agric. Tanganyika Terr. for the twelve months ending 31st March, 1926, p. 33-36. (R.A.M. 6: 398. 1927).
27. Seymour, A. B. 1929. Host index of the fungi of North America. Harvard Univ. Press. Cambridge, Mass.
28. Stephens, J. L. 1952. Pastures for the Coastal Plain of Georgia. Georgia Coastal Plain Exp. Sta. Bull. 27 (revised): 1-56.
29. Suryanarayana, D. 1952. Infection caused by the oospores of Sclerospora graminicola (Sacc.) Schroet. on Pennisetum Typhoides Stapf and Hubbard. Indian Phytopath. 5: 66-75.
30. Young, G. V., C. L. Lefebvre, and A. G. Johnson. 1947. Helminthosporium rostratum on corn, sorghum and pearl millet. Phytopath. 47: 180-183.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA.

CURVULARIA ON GLADIOLUS IN CANADA[1]

J. A. Parmelee[2]

Early in 1953 gladiolus corms of the variety Radiance that had been grown from small corms in 1952 were received from a grower in the Montreal area. The symptoms were unlike those of any of the common diseases usually encountered on corms produced in Canada, although they were not unlike those of hard rot caused by Septoria gladioli Pass.

The lesions were present on the cheeks of the corms and sometimes between the scars of the scale attachments that mark the vascular bundle endings. More often they were astride a scar and bounded by the adjoining upper and lower scars. The slightly sunken lesions were light to dark brown, often with a fawn-coloured centre and a darker border; in outline they were elongated in the direction of the scale scar or sometimes irregularly circular (Figs. 1 and 2). Penetration was slight to moderate (Fig. 3) and many lesions were completely walled off by a layer of periderm.

At first some difficulty was experienced in isolating the organism responsible for the lesions. Freehand sections of the lesions showed that a fungus mycelium was present and that it advanced only slightly beyond the visible necrotic area. Subsequent isolations were made using small pieces taken from the junction of the diseased and healthy tissue and pure cultures of the fungus were obtained. It proved to be a Curvularia sp. (Fig. 7).

In the summer of 1953 spotted gladiolus leaves were submitted by the same grower. After they were placed in a moist chamber conidiophores and conidia of Curvularia were produced on the leaf spots.

This is the first time that the Curvularia disease of gladiolus has been observed in Canada.

In March 1954 a visit was made to the grower who submitted the diseased corms in 1953 and to other growers in the Montreal area. At a farm about 75 miles from the one from which the original material had been submitted, the writer also found corms that showed lesions. Again the variety Radiance was involved and the symptoms were the same, but the affected corms were larger and the lesions were correspondingly larger. Curvularia was isolated from this collection (Fig. 7).

The other grower had observed lesions on his Radiance corms for the past ten years, that is, since he had first started to grow the variety. The first grower made his initial observations on the disease in 1952 when the variety Radiance was affected. In 1953 he observed the characteristic lesions on the corms of other varieties including: Harry Hopkins, Spic and Span, Aotearoa, King Click, Pactolus, Lipstick, and Montpelier.

With inoculum from the variety Radiance it was not possible to infect cured corms other than through stab wounds. Cheek infections (Fig. 4) were obtained by this method. When a spore suspension was placed on unwounded corms only the actively growing roots became diseased (Fig. 5). These observations support Magie's (3) report that seedlings and plants from small corms are very susceptible to infection. Most of the specimens received were small and medium-sized corms.

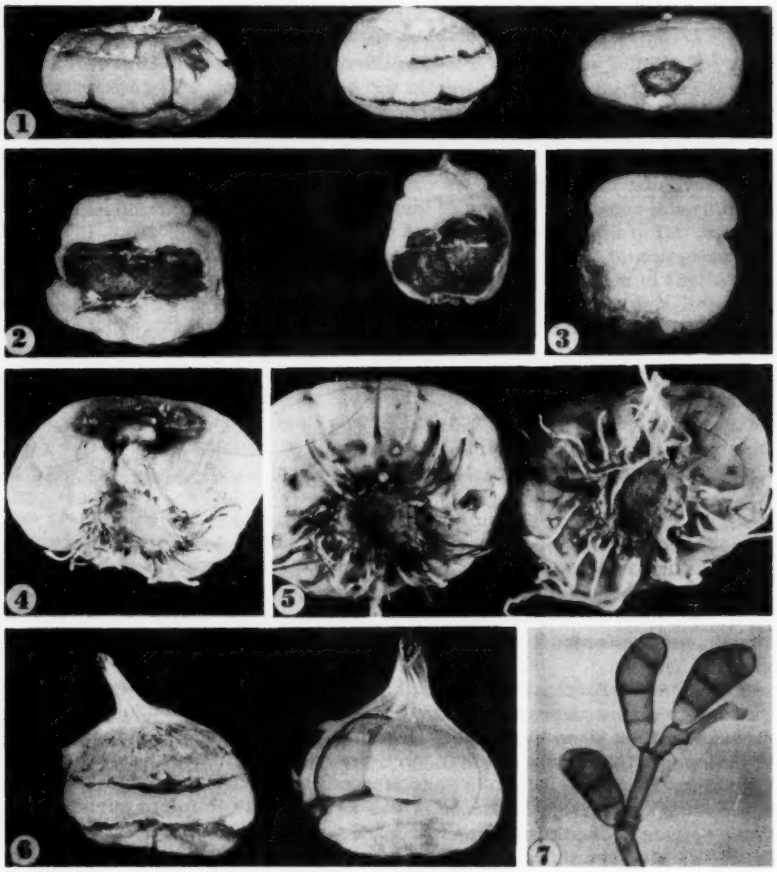
It is presumed that when the perfect state of Curvularia is found it will be similar to other dematiaceous fungi whose known perfect stage is a perithecium or pseudoperithecium. Small, black, globose and erumpent structures which superficially resemble perithecia were found on the scales of the variety Harry Hopkins (Fig. 6). Examination revealed these to be undifferentiated masses of mycelium made up of globose, pigmented cells attached to the substrate by hyphae. A number of these structures placed on moistened filter paper in petri dishes for a week at 10° C produced clumps of conidiophores; and others that had been surface sterilized in a chlorine solution and plated produced typical Curvularia cultures in 50 percent of the plates. In single spore cultures and pairings made from various isolates similar structures have been found along the edge and at the base of the culture dish or tube. Additional study may indicate otherwise, but at present it is thought that the black bodies reported here, and which are entirely different from the erect cylindrical structures recorded by Bonar (1) and Groves and Skolko (2), are structures that assure the survival of the fungus.

The binomial currently in use for the Curvularia on gladiolus is C. lunata (Wakker) Boed. No specific epithet has been attached to the isolate from the Canadian material because the characters used for the differentiation of species, such as spore size, shape and number of septations, are intermediate between C. trifolii (Kauffm.) Boed. and C. lunata.

---

[1]Contribution No. 1391 from the Botany and Plant Pathology Division, Science Service, Department of Agriculture, Canada.

[2]Assistant Mycologist.



FIGURES 1, 2. Curvularia lesions from natural infection (x 1 1/2).

FIGURE 3. Longitudinal section through corm to show depth of penetration (x 1 1/2).

FIGURE 4. Stab wound inoculation (x 3/4).

FIGURE 5. (left) Spore suspension inoculation roots diseased, (right) check roots healthy (x 3/4).

FIGURE 6. Black bodies on corm scales of variety Harry Hopkins (x 1 1/2).

FIGURE 7. Curvularia isolated from variety Radiance (x approximately 383).

Literature Cited

1. Bonar, Lee. 1924. Studies on the biology of Brachysporium trifolii. Amer. Jour. Bot. 11: 3. 123-124.
2. Groves, J. W. and Skolko, A. J. 1945. Notes on seed-borne fungi. III. Curvularia. Can. Jour. Research C, 23: 94-104.
3. Magie, R. O. Botrytis and Curvularia diseases of gladiolus. N.A.G.C. Bulletin 27: 85-90.

BOTANY AND PLANT PATHOLOGY DIVISION, SCIENCE SERVICE, DEPARTMENT OF AGRICULTURE, OTTAWA, CANADA.

CONTROL OF DRY ROT OF GLADIOLUS CORMS IN STORAGE WITH FUNGICIDES APPLIED AFTER HARVEST

Frank A. Haasis[1]

Abstract

Immersion of gladiolus corms (Picardy), shortly after harvest and cleaning, for 5 minutes in Ceresan-M at a concentration of 1 pound to 100 gallons of water practically eliminated development of corm lesions resulting from the dry rot disease, caused by Stromatinia gladioli (Drayt.) Whetz., with no apparent injury to subsequent plant and corm growth.

Dry rot of gladiolus, caused by Stromatinia gladioli (Drayt.) Whetz., though not a serious disease of gladiolus in eastern North Carolina at the present time, is nevertheless frequently observed and in some instances losses have been considerable. These losses have occurred both in the field and in the storage house.

Massey (5, 6) reported failure with preplanting treatments in checking the disease in infected corms by 1) employing long steeps in formalin or bichloride of mercury; 2) dusting corms with sulfur, slaked lime, acid phosphate, or soot; or 3) using these latter materials as soil amendments at planting. Prestorage dips in formalin, or bichloride of mercury; fumigation with formaldehyde; dusting with sulfur, slaked lime, acid phosphate or soot; and high temperature treatments both wet and dry all failed to control the disease.

Drayton (1, 2) reported no control from the use of formalin, bichloride of mercury, Uspulun, or Bayer Compound, employed as preplanting dips for diseased corms or as drenches on infested soil. More recently Forsberg (3) reported failure to control the disease with Arasan, Ceresan-M, Dow F-800, Dow 9B, Dowicide B, Lysol, bichloride of mercury, New Improved Ceresan, Puraturf 177, Semesan, phenyl-mercury-fixtan, Calogreen, Tag 331, and a combination of bichloride of mercury and New Improved Ceresan, when employed as preplanting treatments. Nelson, (7) on the other hand, reported good control of dry rot by immersion of corms in suspensions of calomel or yellow oxide of mercury and also by dusting the corms with these compounds as preplanting treatments. He also stated that storage losses can be reduced by rapid drying of corms following harvest. Gould (4) reported that proper curing procedures, which are adequate for controlling Botrytis rot (Botrytis gladiolorum) in storage, failed to control the dry rot disease adequately. He further reported that a 1-hour preplanting steep of corms in Mersolite-W gave excellent control of dry rot and that a 10- to 60-minute steep in Tersan gave fair to good control. There was no control with 1-hour steep in Natriphene. Although the Mersolite-W treatment was effective in controlling the disease it also caused appreciable plant injury in delayed emergence and reduction in size and weight of harvested corms, whereas Tersan or Natriphene tended to stimulate plant growth.

Recent tests conducted at the Vegetable Research Laboratory near Wilmington, North Carolina resulted in very good control of dry rot by immersing corms in a suspension of Ceresan-M shortly after harvest and fair control by steeping corms in Dow 9B or Dowicide B. A brief report of this work is presented here.

MATERIALS AND METHODS

Picardy corms were harvested on November 28 and 36 hours later cleaned and obviously diseased or injured corms discarded. The apparently healthy corms were mixed thoroughly and divided into samples of 25 corms each. The fungicidal treatments, listed in Table 1, followed immediately with 18 replicate lots comprising each treatment. Thereafter the corms were placed in trays and stored in an unheated but frost-proof shelter until March 5 when disease readings were made.

After storage the variously treated lots of corms were planted in the field in a randomized triple-lattice. Planting and cultural practices were similar to those used by commerical growers except that it was impossible to provide adequate irrigation when needed. The new crop of corms was harvested and yield data recorded.

---

[1] Research Professor of Plant Pathology.

Table 1. Control of dry rot of gladiolus with fungicides applied as prestorage treatments and effect of treatments on subsequent corm production

Chemical	Concentration pounds/100 gal. water	Duration of steep in min.	Mean percent disease as sin-$\sqrt{x}$	Average number of corms harvested per plot	Average weight per per corm after harvest (grams)
Dow 9B -- 50%	3	60	7.6	16.6**	20.72
Dowicide B -- 85%	1 3/4	60	7.7	19.0	20.01
Ceresan-M -- 7.7%	1	5	2.6	18.4	19.28
N. I. Ceresan -- 5%	1	5	13.5	18.2	16.61
Spergon -- 98%	-*	-	15.4	11.8	23.68
None	-	-	16.9	6.8	16.38
L.S.D.'s .05			4.74	-	3.71
.01			6.27	-	4.94

* Corms heavily coated with dust.
** Variance among means due chiefly to losses from Fusarium.

RESULTS

Immersion of gladiolus corms, shortly after harvesting and cleaning, for 5 minutes in Ceresan-M at a concentration of 1 pound to 100 gallons of water practically eliminated development of corm lesions resulting from the dry rot disease (Table 1). Ceresan-M proved significantly better than all other materials tested. Fair control of the disease was also achieved by steeping the corms for 1 hour in equivalent concentrations of zinc trichlorophenate (Dow 9B) or sodium trichlorophenate (Dowicide B); the magnitude of control was equal with these latter chemically related compounds. Neither an immersion of corms in New Improved Ceresan for 5 minutes at a concentration of 1 pound to 100 gallons of water nor a thorough surface coating of corms with Spergon reduced the incidence of disease materially. Throughout the growing period periodical observations revealed no difference in growth performance which could be attributed to treatment except as treatments influenced losses due to Fusarium oxysporum f. gladioli Snyder & Hansen, which is not dealt with here.

After harvest, the corms were allowed to dry for 2 weeks and then weighed. The average corm weight of each lot was determined and analysed statistically in order to detect weight differences attributable to the prestorage treatments. Owing to the planting arrangements the block effects were eliminated. Hence, the means for average corm weight (Table 1) have been adjusted for block effect. On the basis of yield per individual corm, Dow 9B, Dowicide B, Ceresan-M, and Spergon-98% were virtually non-injurious, whereas New Improved Ceresan may have caused injury.

The growing season was excessively dry and thus probably accounts for the inordinately low corm yield.

SUMMARY

It appears that a sanitizing treatment of a Ceresan-M dip applied shortly after corm harvest will effectively reduce storage loss in gladiolus incited by S. gladioli without jeopardizing subsequent corm growth.

Literature Cited

1. Drayton, F. L. 1926. The dry rot disease of gladioli. Phytopath, 16: 757.
2. Drayton, F. L. 1926. The dry rot disease of gladioli. Sci. Agr. 6: 199-209.
3. Forsberg, J. L. 1950. Summary of data on cooperative gladiolus corm treatment experiment for 1949. Third Ann. Rept. of the Spec. Com. of the Amer. Phytopath. Soc. on the coordination of field tests with new fungicidal sprays and dusts. (Mimeographed).
4. Gould, C. J. 1951. Gladiolus dry rot tests in Western Washington. Plant Dis. Reptr. 35: 109.
5. Massey, L. M. 1916. The hard rot disease of gladiolus. New York (Cornell) Agr. Exp. Sta. Bul. 380: 149-181.
6. Massey, L. M. 1928. Dry rot of gladiolus corms. Phytopath. 18: 519-529.
7. Nelson, Ray. 1948. Diseases of gladiolus. Michigan State College Agr. Exp. Sta. Bul. 350: 1-63.

VEGETABLE RESEARCH LABORATORY, CASTLE HAYNE, NORTH CAROLINA, AND PLANT PATHOLOGY LABORATORIES, RALEIGH, NORTH CAROLINA

PROGRESS REPORT OF THE COMMITTEE ON SEED AND PLANT MATERIAL CERTIFICATION AND A REQUEST FOR CONTRIBUTIONS FROM AMERICAN PLANT PATHOLOGISTS

During the last three years, the American Phytopathological Society Committee on Seed and Plant Material Certification has attempted to obtain information for development of pathological standards usable in certification procedures for reduction of seed-borne diseases. The information received was generally insufficient to justify more than a few proposals of changes in pathological standards. The committee believes that much additional information exists and could be utilized if it were made available. This report was prepared primarily to explain the committee program with the hope that our fellow plant pathologists will consider their individual plant disease problems and contribute their recommendations and data to the various commodity sub-committees.

The work project mentioned above was begun in 1950 to determine whether measures for reducing seed-borne diseases were available but were not being utilized in seed and plant material certification. In order that the first project might be completed within a reasonable time, the initial survey was limited to those crops for which uniform standards had been adopted by the International Crop Improvement Association. These crops were alfalfa, beans (garden, field and mung), birdsfoot trefoil, clovers (red, sweet, white and crimson), corn (open pollinated and hybrid), cotton, flax, grass, lespedeza, onion, peas, peanut, small grains (wheat, barley, oats and rye), sorghum, soybeans, tobacco, vetch and watermelon.

A review of the uniform standards of the I.C.I.A.[1] revealed that certification minimum standards had been adopted for only a few diseases in only a few of these crops. Disease standards in force in 1950 were on head and kernel smut on sorghum seed, chemically controllable seed-borne diseases in small grain seeds, bacterial blights, mosaic (common) and anthracnose in field and garden beans and mung beans.

ST. LOUIS MEETING OF THE INTERNATIONAL CROP IMPROVEMENT ASSOCIATION

From the sketchy information received in our survey, only a few definite suggestions about pathological standards could be submitted to the I.C.I.A. at their annual meeting in St. Louis, November 11, 1953. These were as follows:

Field and Garden Beans. That mosaic be specified as Common Bean Mosaic in the standards to distinguish it from yellow bean mosaic and other viruses that are not known to be seed-carried.

Regarding specific disease standards, more stringent requirements are generally agreed as necessary. For bacterial blight, bean common mosaic and anthracnose, the goal should be no tolerance in foundation or registered seed and near zero in certified seed.

An isolation requirement would be helpful in avoiding infection. An isolation up to one mile from other beans would be extremely helpful in foundation and registered seed.

Potato. The potato minimum standards proposed in the I.C.I.A. potato committee report for 1952 are commendable. However, from the experience in Oregon, it was suggested that these standards for foundation seed potatoes had proved to be too lenient, especially for mosaic and leaf roll. A 0.5 percent tolerance in the second inspection could be from tuber perpetuation, and the current season spread may not show, if from late infection. This could result in up to 25 percent infection in the tubers. Mosaic is even worse than leaf roll for symptoms not showing from late infections. For example, an Oregon sample once showed no field symptoms in the second inspection, thus had a zero reading, but the samples showed up to 50 percent tuber infection with mosaic.

To help reduce seed-borne virus diseases, it was suggested that an isolation distance for foundation fields of 500 yards from uncertified potatoes be established unless the latter potatoes also meet the same standards.

Some of the other information obtained by the survey was discussed at St. Louis, because it indicated the trends of possible future proposals.

---

[1] Taken from Publication No. 17, Minimum Seed Certification Standards, December, 1950. Published by International Crop Improvement Association.

Probably the most profitable outcome of the St. Louis meeting was the agreement on how to coordinate the efforts of the two committees studying pathological standards. For our A.P.S. Committee, we have established commodity sub-committees to distribute the work load. Fortunately, the chairmen of these sub-committees are specialists in the diseases of the crops to which they have been assigned and can give effective leadership to their sub-committees, which are as follows:

Chairman	Commodity Sub-Committees
C. H. Arndt Dept. of Botany & Bacteriology Clemson Agr. Experiment Station Clemson, South Carolina	Certification of peanut, tobacco, sugar cane, cotton and other Southern crops.
Wayne Bever Agronomy Dept. University of Illinois Urbana, Illinois	Certification of small grains, corn, sorghum, flax seed.
John T. Middleton Plant Pathology Dept. University of California Riverside, California	Certification of vegetable crops (peas, beans, onions, watermelons, etc.)
L. C. Cochran Citrus Experiment Station Riverside, California	Certification of fruit crops.
J. R. Hardison Oregon Agr. Experiment Station Corvallis, Oregon	Certification of forage crops.
Reiner Bonde Plant Pathology Dept. Maine Agr. Experiment Station Orono, Maine	Certification of potatoes.
Kenneth Baker Department of Plant Pathology University of California Los Angeles 34, California	Certification of ornamental crops.
W. H. Wheeler Nursery Stock Section Plant Quarantine Branch Washington 25, D. C.	Measure to protect against introduction of foreign plant diseases in imported seed and plant materials.

It is our plan that problems pertaining to pathological standards of particular crops will be submitted to the appropriate sub-committees for study. We hope to publish periodically descriptions of the pressing problems in the Plant Disease Reporter with a request for contributions. Proposed new or revised standards can be submitted for approval by the membership of the American Phytopathological Society and then submitted to the I.C.I.A. for consideration.

COOPERATION OF THE INTERNATIONAL CROP IMPROVEMENT ASSOCIATION

The A.P.S. Committee program was well received at the I.C.I.A. meeting. The interest and endorsement of that group was expressed in the 1953 Report of the Committee on Pathological Standards, part of which is quoted as follows: "The work of these sub-committees (A.P.S.) should eventually result in complete analyses of the significant seed-borne diseases and may result in workable pathological standards for each crop."

The general procedure outlined for the cooperative action of these two committees includes the following:

1. The Committee on Seed and Plant Material Certification of the A.P.S. will annually receive and advertise the problems submitted by publication in the Plant Disease Reporter in order that all pathologists will have an opportunity to know of the problems and provide information and make such recommendations as seem applicable from their knowledge and experience.
2. The appropriate sub-committee will receive and consider all such recommendations and information and report back to the general A.P.S. Committee.
3. The general A.P.S. committee will report annually to the I.C.I.A. Committee on Pathological Standards.
4. The I.C.I.A. Committee on Pathological Standards will evaluate all information so presented and pass on to the commodity committees such information and recommendations as seem usable.
5. I.C.I.A. Commodity Committee reports are to be provided each year to the A.P.S. Committee members to keep them informed of actions of these groups.

"The I.C.I.A. Committee on Pathological Standards urges the support of all commodity committees to the extent that all pathological problems considered by the committee be reported to the proper sub-committee from the A.P.S. These reports may be sent directly to the sub-committee chairmen listed above, or may be sent to Dr. Hardison or may be routed through your state pathologists to the committee.

"It is recommended by your Committee on Pathological Standards that the I.C.I.A. endorse the work as outlined above, since it appears to the committee that this procedure offers an opportunity for seed certification interests to draw upon a large source of information and assistance in pathology which in the past has not been readily available to our organization. It is the feeling of your committee that this action can and will lead to a continuing source of very helpful material in building pathological standards for crops covered by our commodity committees."

RECENT CHANGES IN PATHOLOGICAL STANDARDS

Since 1950, several committees of the I.C.I.A. have studied pathological standards, and definite proposals have been made. The actions on minimum pathological standards as proposed or under consideration by the I.C.I.A. Commodity Committees from 1950 through 1953 are as follows:*

COTTON COMMITTEE

Changes and additions recommended, 1953. Section IV. FIELD STANDARDS, Subsection 2. Specific Requirements.

3. As for diseases follow same provisions as for weeds, i.e. "objectionable diseases" etc. (Thus no specific requirements are established except to specify "Precaution should be taken to avoid the possibility of gathering objectionable diseases with cotton when the seed crop is harvested mechanically.")

I.C.I.A. POTATO COMMITTEE

Minimum standards for certified seed potatoes on a national level were first proposed in 1951. These were as follows:

II. LAND REQUIREMENTS
   1. Isolation. All fields producing certified seed shall be at least 50 feet from any non-certified potato fields, unless such fields meet the same certification standards.

III. FIELD INSPECTION
   1. At least two field inspections shall be made.
   2. Disease tolerances for field inspection.

* Published by permission of the International Crop Improvement Association.

	First Inspection	Second Inspection
Spindle Tuber	2%	2%
Mosaic	3%	2%
Leaf Roll	3%	2%
Other Virus	3%	2%
Total Virus Diseases	5%	3%
Ring Rot	None	None
Varietal Mixture	1.0%	0.5%

Adoption of the above proposed minimum standards without change as the official minimum standards for certified seed potatoes was recommended at the 1952 meeting. Also, in 1952, tentative minimum standards were proposed for foundation seed potatoes as follows:

MINIMUM STANDARDS FOR FOUNDATION SEED POTATOES, proposed in 1952.

Land Requirements

1. Isolation. All fields producing foundation seed shall be at least 200 feet from any other potato fields.

Field Inspection

1. At least two field inpsections shall be made
2. Disease tolerances for field inspection:

	1st Inspection	2nd Inspection
Spindle Tuber	1.0%	0.5%
Mosaic	1.0%	0.5%
Leaf Roll	1.0%	0.5%
Other Virus	1.0%	0.5%
Total Virus Diseases	1.0%	0.5%
Ring Rot	None	None
Variety Mixture	0.25%	None

"The committee also recommends that a winter testing program be adopted to reveal the amount of late infection of virus diseases in foundation seed."

In the 1953 report, it was stated that, "The committee found that to make the standards more strict would mean that nearly one-half of the states reporting standards for foundation seed potatoes would have to revise their standards. The committee felt that since these standards are only minimum standards, and that any state can decrease the tolerances as much as they find practical, it would be unwise to change them at this time.

"Therefore, the potato committee recommends that the proposed Minimum Standards for Foundation Seed Potatoes as published in the 1952 Annual Report of the International Crop Improvement Association be adopted as the official standards for Foundation Seed Potatoes, and that these standards be made a part of the minimum standards for Certified Seed Potatoes published in the 1951 Annual Report of the ICIA."

Small Grain

At the 1953 meeting at St. Louis, sub-committees on wheat, oats, barley and rye were appointed. Agronomic requirements for each of these small grain crops of wheat, oats, barley and rye are to be studied separately, because each crop presents a different problem. It appears to be opportune for plant pathologists to re-examine the small grain standards for possible modifications.

VEGETABLE AND TRUCK CROPS COMMITTEE

Following amendments recommended:

I. TOMATO STANDARDS

IV. FIELD STANDARDS

2. Specific Requirements

(1) Disease	Foundation	Registered	Certified
Bacterial Canker	-	-	Trace*
Streak	To be specified by the certifying agency		
Fusarium Wilt	To be specified by the certifying agency		
Phytophthora Foot Rot	To be specified by the certifying agency		
Septoria Foliage Blight	To be specified by the certifying agency		

*Less than 0.1%

PATHOLOGICAL STANDARDS COMMITTEE

In 1951 the Committee on Pathological Standards recommended to the member associations of the International Crop Improvement Association that all breeder and foundation seed increased for the production of registered or certified seed be so treated with chemical and/or other seed treatments so as to hold to a minimum any disease or diseases which can be controlled.

DISCUSSION

Cooperation with the I.C.I.A. offers one of the best opportunities for controlling seed-borne diseases. In 1953 this body had committees for each of the following commodities: Alfalfa and Trefoil; Field and Garden Beans; Clover; Corn; Cotton; Flax; Grass; Lespedeza; Millet; Other Crops; Potato; Rice; Small Grain; Sorghum; Soybeans; Tobacco; Vegetable and Truck Crops incl. Melons; and Vetch and also a Committee on Pathological Standards.

Dr. R. J. Haskell has served as a member of the Pathological Standards Committee of the I.C.I.A., retiring in 1952. Other plant pathologists have served either informally, as guest speakers or as members of commodity committees. In 1953, three members of Pathological Standards Committee were plant pathologists: J. H. Craigie, Canada, C. O. Johnston, Kansas; and J. R. Hardison, Oregon. The A.P.S. Committee was represented at the 1953 St. Louis meetings by Wayne Bever and Hardison.

Pathologists, because of their training and the nature of their work, are best qualified to consider certification regulations as measures of disease control. In fact, a pathologist during the investigation of a disease, may advisedly consider the application of certification standards as an integral part of control and thus obtain specific information for certification during the initial investigation. Other pathologists, less familiar with the disease and crop concerned would be in a less favorable position to formulate such standards. These considerations indicate that we cannot expect certification officials to progress in this phase of disease control without the help of plant pathologists. There undoubtedly exist methods for reducing seed-borne diseases that could be used to maintain the effectiveness of the initial protection provided by seed treatment. A few of the measures that seem to have been neglected are as follows:

LAND REQUIREMENTS

1. CROP ROTATION AND PREVIOUS CROP HISTORY

Many diseases are controlled or greatly reduced by crop rotation, and freedom from susceptible crops for the necessary number of years could be considered as a requirement for eligibility of land to produce certified seed crops. Where weed hosts are important in perpetuating a disease, the necessary "clean" crop rotation might be secured by growing non-susceptible crops plus selective weed sprays or cultivated crops for a specified number of years.

Example A. (From Mississippi regulations):

Watermelons may follow corn, cotton, small grains and other grasses, or crops not commonly susceptible to Southern Blight. Certain vegetable crops transmit Southern

Blight to melons.

Example B. (From North Carolina regulations):

"Sweet potatoes should not be planted on land:
1. that has produced sweet potatoes for at least 3 years.
2. that has received manure or sweet potato residue during the last 3 years.
3. that is subject to drainage from fields that are now growing or have grown sweet potatoes for a period of three years."

2. ISOLATION

A minimum isolation distance requirement has been established for most certified seed crops to protect genetic purity, but this means of protecting against disease contamination seems to have been neglected almost completely. A minimum distance between certified fields and other fields of the same crop has been established for seed potatoes to avoid infection by virus diseases. It appears desirable that similar requirements be stipulated for beans. The minimum distance from other fields of infected wheat necessary to avoid loose smut contamination in foundation and registered classes of seed wheat needs to be determined. North Carolina and New Jersey regulations each specify a different minimum distance from other fields with a specified amount of infestation.

Possibly a minimum distance from weed hosts and uncultivated areas that furnish inoculum should be specified. In the case of insect vectors, perhaps direction of prevailing winds should be a factor in determining isolation.

3. FIELD SANITATION

In addition to previous crop history of land, disinfestation of land growing certified seed crops may be desirable particularly for perennial crops. Some diseases are effectively controlled by burning crop residues after harvest. Burning, removal of crop residues, deep plowing and other methods of field sanitation might well be explored as possible requirements in the production of certified seeds.

4. SEED INSPECTION STANDARDS

Disinfestation of Seeds.

The disinfestation of crop seeds can sometimes be accomplished by special cleaning equipment. A good example is the removal of Sclerotinia sclerotia from legume and certain vegetable seeds by specific gravity separators using air flotation. Disease material, such as sclerotia, are now classified as "inert." Studies are needed to determine what inert disease material and infected seeds can be removed from seed crops based on the differences in specific gravity or otherwise. If such material can be completely removed readily by available machinery, perhaps we should consider low or zero tolerances in seed inspection standards.

5. TREATMENTS OF THE GROWING CROP

Fungicidal Sprays and Dusts

Few, if any, applications of fungicidal sprays or dusts are suggested or required in seed certification regulations. Perhaps we should review the possibilities of reducing seed infections by fungicide applications. If leaf and stem infections can be controlled, the reduction in inoculum should in some cases reduce the amount of seed infection.

6. SEED TREATMENT

Seed treatment, even now, is not a requirement in some seed crops where it is known to be helpful. We do need to distinguish between treatment of seed used to produce certified seed crops and treatment of certified seed sold to the final consumer. It is sometimes suggested that seed treatment should be compulsory to protect against soil-borne diseases which may or may not be seed-borne, e.g. seedling blights of grasses. Without seed carriage, a

QUESTIONNAIRE ON PATHOLOGICAL STANDARDS FOR USE IN CERTIFICATION

Name of Crop:

Should treatment of seed to plant seed production fields be required? If so, specify treatment.

LAND REQUIREMENTS: (Rotation, Field Sanitation, etc.)

FIELD INSPECTION: (time, number)

FIELD STANDARDS:

Isolation: (distance)

Specific Requirements Disease Factor	Maximum permitted in each class Breeders - Foundation - Registered - Certified
Total Diseases	

SEED STANDARDS: Disease Factor	Maximum for each class Breeders - Foundation - Registered - Certified

Seed Treatment: Is there an alternative and effective seed treatment that can be substituted for disease freedom? If so, specify disease and treatment.

General: Are present uniform standards satisfactory? Yes_____ No_____

Remarks:

Your Name_____ Address_____

(Please use reverse side for additional information)

required seed treatment would not be in harmony with the usual concept of seed certification practices but appears to be more of a problem of educating the consumer to the desirability of seed treatment in problem areas.

A possible over-emphasis of seed treatment was described by Dr. W. B. Allington in correspondence, "certainly more research is needed to develop measures for control of seed-borne diseases. Perhaps relatively too much emphasis is being placed on chemical seed treatment and not enough on other possible means of avoiding seed transmission."

There are some fundamental considerations in applying disease standards. Foremost appears to be the question of recognizing the disease in field or seed inspection. If the standards are applied too rigorously, there may not be any seed left to certify. It may be desirable to raise the minimum disease standards gradually so that seed growers can be educated during the period of adjustment.

There are many crops for which a disease standard would be highly desirable, but no usable tests for detection of disease are available to seed analysts. Here, research by the pathologist investigating the disease could contribute materially to the problem.

Above all, pathological standards must be workable in certification procedures. Certification specialists in most states can help devise feasible standards from their experience in certification practices.

Generally speaking, new proposals for disease standards will be more readily adopted, if the requirements can be easily met. Land requirements ought to be the most attractive, because they prevent wasteful expenses on a crop that might later be rejected. Cultural practices should also meet with favor, because they can often be accomplished with equipment on hand with a minimum of expense and inconvenience. Disinfestation of seed when possible by seed cleaning machinery also will not cause great hardships.

NEEDED INFORMATION FOR DEVELOPMENT OF PATHOLOGICAL STANDARDS

In preparing contributions, your attention is directed to the existing minimum standards and the discussion on new ideas for certification measures. We would appreciate your evaluation of these existing pathological standards and your opinion as to whether the tolerances are too lenient, too stringent, or satisfactory. Also, we want your suggestions about additional seed-borne diseases that should be considered and, if possible, any measures you wish to propose to reduce specific diseases.

Special summaries of state standards on cereal crops, southern crops and other commodities probably will be presented by the various sub-committees at a later date. When available, these reports will furnish additional material for your evaluation.

The material presented here has been concerned primarily with vegetable and field crops. We would like to remind readers that part of our committee efforts are now devoted to two additional crop groups, FRUIT CROPS, AND ORNAMENTALS, and two of our subcommittees headed by L. C. Cochran and Kenneth Baker would appreciate similar information for these crops.

For your convenience in preparing contributions and our convenience in tabulating the information, your committee suggests use of the accompanying questionnaire whenever possible. Additional copies of this questionnaire are available on request from any member of this committee.

A SPECIAL REQUEST FOR PARTICIPATION BY PLANT PATHOLOGISTS

With the endorsement of our program by the International Crop Improvement Association and expressed offers of cooperation and assistance, it appears that plant pathologists have an excellent opportunity to contribute effectively to the program for reducing seed-borne diseases. Your committee hopes for wide participation from fellow pathologists to fulfill our apparent obligation. Plant pathologists are invited to contribute their data to the various commodity sub-committee chairmen. Any ideas on how to reduce specific seed-borne diseases and how to improve our overall program will be welcomed.

RESPECTFULLY SUBMITTED,

C. H. Arndt	K. F. Baker	L. C. Cochran	W. H. Wheeler
W. M. Bever	Reiner Bonde	John T. Middleton	John R. Hardison, Chairman

COMMITTEE ON SEED AND PLANT MATERIAL CERTIFICATION
AMERICAN PHYTOPATHOLOGICAL SOCIETY

UNUSUAL RECORDS AND OTHER BRIEF NOTES ON PLANT DISEASES

SUNBURNING OF WATERMELON REDUCED BY USE OF LIME PASTE

By G. K. Parris

In 1947 the writer and Mr. Loren H. Stover reported excellent control of sunburn of maturing watermelons in Florida by the use of a lime paste[1]. Since moving to Mississippi, numerous requests for this paste, its formula and method of application, have been received. The original description of the simple procedure is published in the relatively obscure journal cited; it is repeated here so that it may be more easily available to interested persons.

"Sunburn can be reduced by the use of a lime paste which is painted on the maturing melons. A solution is made of 3 to 5 pounds of hydrated lime in 2 gallons of water, well mixed, and applied to the exposed surface of the melon by means of a rag swab on a handle, or with a broad, soft, paint brush. A 4 to 5 inch wide brush is excellent. To make the lime paste adhere well, the melon surface should be brushed back and forth about four times. Once dried, the lime sticks and lasts well and light showers of rain seem to reduce it but slightly; heavy showers do wash off some lime and the paste may have to be renewed. If a second application is necessary, a single stroke with the brush will suffice. Once the discoloration and loss of color, usually associated with sunburning of watermelon fruit, have started, the lime paste will not stop it if applied, probably because internal breakdown of the flesh due to the heat has already started.

"Different methods of removing the lime, which is somewhat unsightly to buyers and the public, have been tried, but the quickest and most practical is to rub off the lime with a moistened rag.

"In 1946 tests with the variety Blacklee where no lime was applied 49 out of 65 melons sunburned; where lime was applied to companion melons in the same field only 15 out of 60 sunburned. If the lime content was reduced to 1 pound per 2 gallons of water the number of sunburned melons jumped to 42 out of 60, almost the same as where no lime was used. The use of lime paste will not eliminate sunburn in watermelons, but it does reduce losses, and the cost is little more than the cost of the labor to apply it".

The writer has seen several references in the literature, subsequent to 1947, to the value of lime paste for reducing losses due to sunburn in watermelons. The idea was not original with him when he was associated with the Watermelon Laboratory at Leesburg; it was an idea that seemed to be perennially appearing, but no one had actually put it to a real test until 1946.

STATE PLANT BOARD OF MISSISSIPPI, STATE COLLEGE, MISSISSIPPI

[1]Parris, G. K. and Loren H. Stover. "Suntan lotion" for watermelons found. Florida Grower 55 (Oct.): 27. 1947.

ROOT DISEASES OF COTTON OBSERVED IN MEXICO AND CALIFORNIA

By John T. Presley

Typical mycelial strands of Phymatotrichum omnivorum were found on the roots of dead cotton plants at the winter increase garden, Iguala, Mexico, in October 1953. Several plants in the species garden were killed by the fungus: among them were Gossypium arboreum, G. robinsonii, G. anomalum, and G. sturtii. The plants of G. robinsonii appeared to be most susceptible since all plants of that species in the garden were killed. Occasional plants of American Upland Cotton (G. hirsutum) in a field planting were also killed but there were no typical "dead spots" in the planting such as occur in the Southwestern United States where the disease is prevalent.

Thielaviopsis root rot or "internal collar rot" caused by Thielaviopsis basicola was observed in cotton growing at the Shafter, California Field Station in the fall of 1953. The symptoms were typical of those occurring on mature plants. There was considerable swelling of the plant at and just below the ground line and the central cylinder was reddish-brown or mahogany colored.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, BELTSVILLE, MARYLAND

DECIDUOUS OAKS IN LEAF UNDER GREENHOUSE CONDITIONS THROUGHOUT THE WINTER 1953-1954

By Bert M. Zuckerman and P. F. Hoffman

In an attempt to have native oaks in foliage during the winter for oak wilt studies in the greenhouse, 100 red oaks (Quercus borealis maxima (Marsh.) Ashe), including 50 three-year-old and 50 four-year-old trees, were obtained from the Cook County Forest Preserve nursery in April, 1953, prior to bud break. The roots of the three-year-old trees were covered with moist sphagnum, after which the trees were securely wrapped in one bundle with waterproof paper. The four-year-old trees were treated in the same manner. The two bundles were then stored at 32° F until late September, 1953, when they were removed from storage, potted, and placed in the greenhouse. By the end of October, 1953, 26 (52 percent) of the three-year-old oaks had leafed out, but only three (6 percent) of the four-year-old oaks survived. The surviving oaks were useful for oak wilt studies because they remained in leaf and continued to grow throughout the winter months of 1953-1954.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, NATURAL HISTORY SURVEY, URBANA, ILLINOIS

SCLEROTIUM ROLFSII ON PHILODENDRON CORDATUM IN SOUTHERN CALIFORNIA

By Alex M. French and Carl W. Nichols[1]

Sclerotium rolfsii Sacc. (Pellicularia rolfsii (Sacc.) West) has been known to be the incitant of a serious disease of sugar beets in California since 1931[2]. It has also been found infecting many other hosts in California and other States[3, 4]. The purpose of this paper is to report the occurrence of this fungus on Philodendron cordatum Kunth grown under glass in southern California.

In December, 1953, extensive losses were observed in a bed of rooted cuttings of Philodendron cordatum in a Los Angeles wholesale greenhouse. The leaves of the affected plants were yellowed and flaccid, and many leaves dropped to the surface of the bed. Stems of affected plants were watersoaked and rotted above and below the soil surface. Mycelia and sclerotia typical of Sclerotium rolfsii were observed on the stems, and on the undersides of irregular necrotic spots on many of the fallen leaves. Although about 80 percent of the plants in the infested bed had been killed by the fungus, in immediately adjacent beds similar plants were completely unaffected, and no evidence of the fungus could be found. The source of the fungus could not be determined with certainty, but was presumed to have been the leaf mold incorporated in the soil mix, or the original cuttings themselves. Sources of both differed from those of the adjacent beds.

This appears to be the first recorded instance of infection of Philodendron by Sclerotium rolfsii.

BUREAU OF PLANT PATHOLOGY, CALIFORNIA DEPARTMENT OF AGRICULTURE, RIVERSIDE AND LOS ANGELES, CALIFORNIA.

---

[1] Associate Plant Pathologists, California Department of Agriculture, Riverside and Los Angeles, California.

[2] Leach, L. D., and A. E. Davey. 1935. Toxicity of low concentrations of ammonia to mycelium and sclerotia of Sclerotium rolfsii. Phytopath. 25: 957-959.

[3] Weber, F. G. 1931. Blight of carrots caused by Sclerotium rolfsii with geographic distribution and host range of the fungus. Phytopath. 21: 1129-1140.

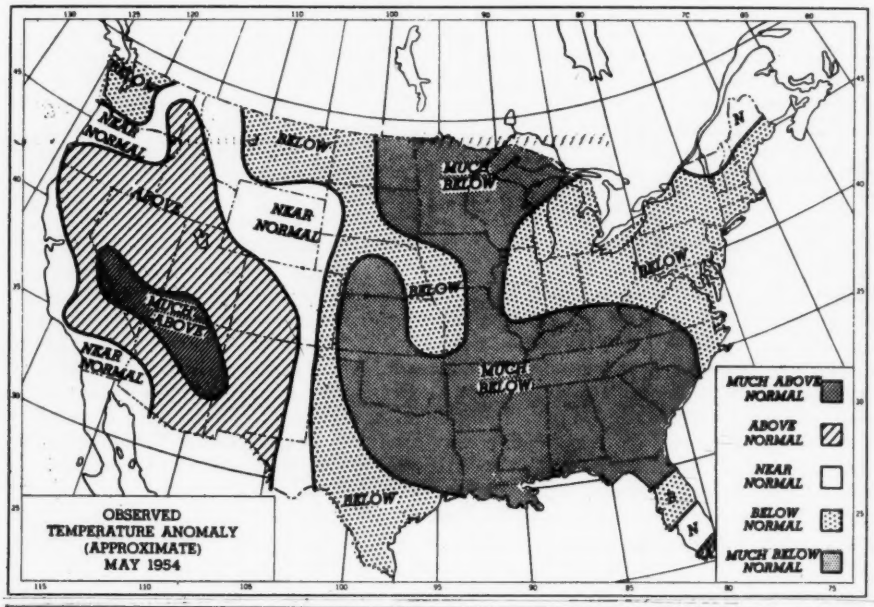
[4] Weiss, Freeman. 1950-1953. Index of plant diseases in the United States. U.S. Dept. of Agric., Plant Disease Survey Special Publication.

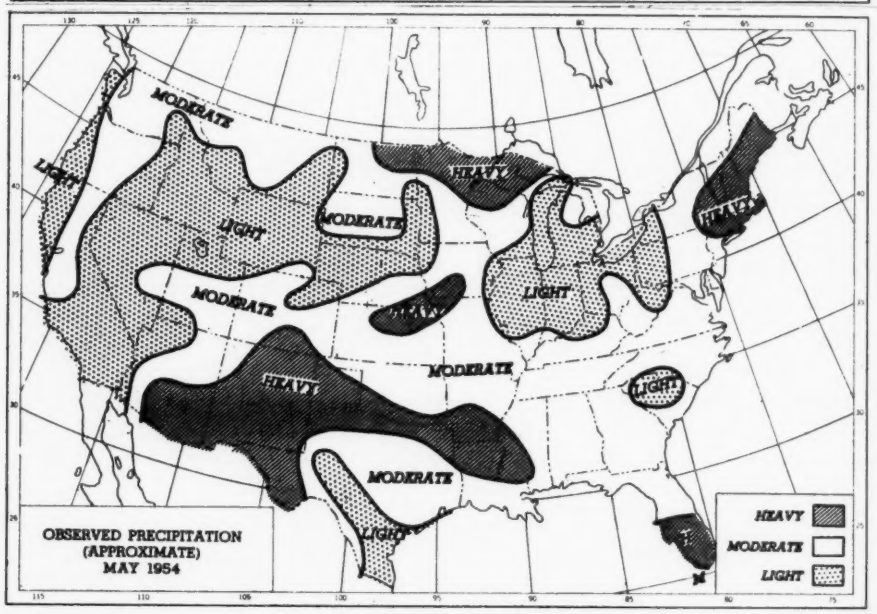
ANNOUNCEMENTS

"MANEB": The Interdepartmental Committee on Pest Control announces that the word "maneb" has been selected as a coined name for the fungicidal chemical manganese ethylenebisdithiocarbamate, previously known by its chemical name, and also as manganese EBD, MnEBD, and sometimes as MEB.

This name thus joins the list of common names for fungicides, which includes zineb, nabam, ferbam, ziram, thiram, chloranil, captan, dichlone, and glyodin.

RESULTS OF THE 1953 FUNGICIDE TESTS: "Results of 1953 Fungicide Tests", reprinted from a series of articles in Agricultural Chemicals January through March 1954 may be purchased in bound and covered form for $1.00 per copy by sending orders with remittance to Dr. W. D. Mills, Department of Plant Pathology, Department of Agriculture, Cornell University, Ithaca, New York. The publication of these "Results" is under the sponsorship of the American Phytopathological Society. It is a continuation of the publication of "Results" formerly provided through a Supplement of the Plant Disease Reporter. The underwriting of this project was accomplished through contributions from many commercial companies identified with the pesticide industry. The Temporary Advisory Committee on Collecting and Disseminating Data on New Fungicide Tests of the American Phytopathological Society arranged for the recent publication of these data and the establishment of a program for annual publications of Fungicide Test Results in the future. Dr. D. A. Roberts, Department of Plant Pathology, Cornell University, Ithaca, New York, will be in charge of this project during the current year.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

and
past
and
sses
the
cted
ime
only
ies,
ime